Suid-Afrikaanse Tydskrif vir Geneeskunde : South African Medical Journal

Kaapstad, 24 Junie 1961 Deel 35 No 25 Volume 35 Cape Town, 24 June 1961

THE TURKISH EPIDEMIC OF PORPHYRIA

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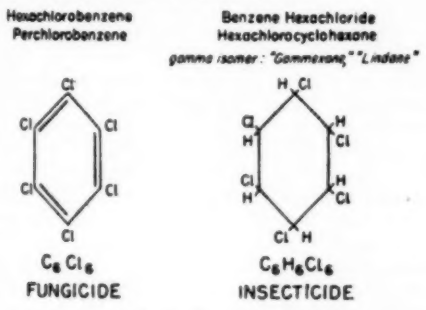
In 1955 Dr. Cihad Cam, who is the Director of the skin clinic in Diyarbakir in Eastern Turkey, found that he was seeing a large number of children with sores and blisters on the face and on the back of their hands. These children had dark pigmented skins and great hairiness of their faces. The urine of the children was reddish-brown in colour and when Dr. Cam examined the urine in ultraviolet light, using a Wood's filter, it gave a brilliant red fluorescence. He realized these children had porphyria. He had not seen children with porphyria before 1955, but in that year and in each subsequent year he saw many hundreds of affected children[1,2] (Fig. 1).

This epidemic aroused great interest and concern in Turkey. Children were admitted for further study under the care of Dr. Joseph Wray to the Hacettepe Children's Hospital in Ankara and also to a hospital in Istanbul. Prof. Cecil Watson of Minneapolis, renowned for his porphyria research, sent one of his outstanding research assistants to assist in the biochemical investigation of the children, and Dr. Rudi Schmid, an American expert on porphyria, visited Turkey and made a report on the epidemic.[3]

The Rôle of Hexachlorobenzene

Dr. Cam, who had taken the dietary history of hundreds of children with porphyria, had made a startling discovery. The peasants in the Eastern part of Turkey are extremely poor and their staple diet is bread. However, it is not possible to obtain good wheat crops unless the seed wheat is treated with a fungicide. In particular, the seed wheat was destroyed by the fungus *Tilletia tritici*. In the past the seed wheat had been treated with mercurous and mercuric chloride which was not very effective against the fungus. In 1954 some of the seed wheat was treated for the first time with 0·2% 'chloroble' or 'surmesan A' which contains the fungicide hexachlorobenzene. This fungicide must not be confused with the insecticide benzene hexachloride:



Chemical Structures of Hexachlorobenzene and Benzene Hexachloride.

Fig. 1 See text

The hexachlorobenzene-treated seed wheat was first issued in 1954 in the Urfa Province, and in that year the first cases of porphyria appeared in that Province. In 1955 the treated wheat was also issued in the Provinces of Diyarbakir and Mardin. Dr. Cam found the affected children had been eating bread made from the wheat which had been issued to the peasants for planting; he quite rightly suspected that the seed wheat was responsible for the epidemic.

The announcement of his views caused a considerable outcry in the newspapers. The matter was complicated because the Ministry of Agriculture believed, with good reason, that unless the wheat was treated with the hexachlorobenzene the crops would be very poor. The Health Ministry, on the other hand, wished to stop the use of the new fungicide. The problem has not yet been resolved and the hexachlorobenzene-treated wheat is still issued, with due warning that it should

not be eaten. Nevertheless much of the wheat is still milled into flour and every year many hundreds of the children develop the symptoms of porphyria. Dr. Cam estimates that about 5,000 children have serious symptoms of porphyria and no doubt many thousands more are mildly affected.

Forms of Cutaneous Porphyria

Three forms of cutaneous porphyria have already been described: 1. A very rare form of porphyria, which causes cutaneous symptoms in infancy, is known as congenital, or erythropoietic porphyria; less than 70 cases have been described in the world literature. This rare disorder is inherited as a Mendelian recessive character, it is associated with anaemia and an enlarged spleen, and the porphyrin production is in the bone marrow. 2. In South Africa many Afrikaners are affected by an inherited form of porphyria that often causes blistering of the exposed skin and some increased pigmentation and hairiness in adults, but seldom in children. This is a Mendelian dominant form of porphyria, porphyria variegata; all the South African cases trace back to one ancestor who married at the Cape in 1688.[4,5]

3. A cutaneous form of porphyria which is not inherited, and which causes similar cutaneous symptoms in middle life, porphyria cutanea tarda, or symptomatic cutaneous porphyria, is also found sporadically. It is often associated with the excessive use of alcohol. This symptomatic type of porphyria is frequently seen in the Bantu, especially if they are accustomed to drinking an excessive amount of the native drink, skokiaan.[6]

The Turkish Epidemic

I visited Turkey in 1960 to see the affected children. Unlike congenital porphyria the symptoms did not start in infancy. The Turkish children developed much greater pigmentation of the skin and hairiness of the face than occurred either in porphyria variegata or in the sporadic cases of symptomatic porphyria in adults. In the villages, several members of the same family were frequently affected. They are called the 'monkey children' by the peasants because of the pigmentation and hairiness of the face. Besides blisters and sores on their hands and faces and the dark pigmentation and hairiness, these children usually had enlarged livers, and liver-function tests showed marked impairment of liver function. They did not have splenomegaly and were usually not anaemic. They were generally undersized and had apparently been undernourished before they developed porphyria. Their dark-red or brown urine contained great quantities of porphyrin, but no increase of the porphyrin precursor, porphobilinogen. There was little or no increase in the porphyrin excretion in the faeces. Analysis of the urine and stool porphyrin gave results very similar to those found in the Bantu cases of cutaneous porphyria.[7,8]

Most of the children with cutaneous symptoms were aged between 5 and 15. In some affected families, babies at their mother's breast developed pigmented patches on the skin rather like a ringworm or fungus infection. These babies did not have a sensitive skin that abraded or blistered, nor did they excrete increased quantities of porphyrin in the urine. No fungus has been recovered from their skin lesions, which are not at all like cutaneous porphyria. It is possible that some of the hexachlorobenzene is absorbed *via* the mother's milk and that in babies it causes a direct skin reaction.

This epidemic is particularly tragic because the children who were first affected in 1956 are still affected today, although they have not eaten bread made from wheat treated with the fungicide since the diagnosis was made. Their sores tend to heal during the winter months and break out again during the summer months, and some of these children have now contractures of their hands and some have osteomyelitis of the bones of the hands and atrophy of the terminal digits. Others have marked scarring of the face. The children who have been treated in hospital on a high-protein diet still show symptoms although they have improved. It appears that once the liver has been so damaged that there is serious disturbance in the porphyrin metabolism, improvement is possible, but relapse occurs very easily and in many cases symptoms persist.

Dr. Schmid has fed rats with the fungicide-treated wheat and they also developed cutaneous porphyria and excreted large amounts of uroporphyrin and coproporphyrin in the urine. The children in the Turkish epidemic did not develop abdominal pain or neurological symptoms, such as occurs in acute porphyria variegata or intermittent acute porphyria, and they did not excrete porphobilinogen in the urine—the porphyrin precursor that is found in the urine in intermittent acute porphyria and in porphyria variegata during an acute attack. In fact the syndrome affecting the children in Turkey is very similar to that affecting the Bantu of South Africa and the sporadic cases of porphyria cutanea tarda, except that children are affected and not adults, and the pigmentation of the skin and the hirsutism is greater than has been seen before—a truly remarkable phenomenon.

The difficulty in distinguishing between the treated and untreated grain makes it hard to discover how much of the seed grain is used for food. It is also difficult to find out how long the patient has been eating the toxic wheat. However, except for a few sporadic cases, all the children have been affected in the Provinces of Diyarbakir, Mardin and Urfa. Most of the wheat has been sold in the country areas and the affected children are mainly from the villages and farms and not from the towns.

It appears likely that the symptoms of cutaneous porphyria are so severe in these children because they already suffered from malnutrition, in particular shortage of sufficient protein, and, as a result, had poor liver function. The Ministry of Agriculture considers that it is not feasible to stop the distribution of the hexachlorobenzene-treated seed wheat on account of the danger of famine. However, it appears to be extremely difficult to prevent the wheat from reaching the merchants and millers. No effective measures have yet been found to stop the epidemic. My suggestion was that the seed wheat should be stained with a dye, perhaps methylene blue, and a small proportion of it crushed. Then, if it was used for making flour, the dye would pass into the flour and would colour the bread. If this was done the wheat would be easy to trace and it would not be possible to use the wheat for making bread by mistake. It is most important that urgent action should be taken because otherwise, in the affected provinces, a large number of the future generation are going to be permanently handicapped.

COMMENT

Poverty and ignorance is the root of the problem. The peasants in Eastern Turkey are living on a diet which is

extremely low in protein, the children usually getting no meat and very little milk. The protein of the diet could be increased cheaply and effectively by adding protein to the flour that is used for making bread. For instance, soya-bean protein and protein from fish which is readily available from the coastal areas. The fortified flour could then be sold to the very poor Turkish and Kurdish peasants in Eastern Turkey.

The Bantu of Africa also eat a diet which is lacking in sufficient protein. The Government of South Africa has done a great deal to counteract protein deficiency in the Bantu by adding protein to wholemeal flour. This can apparently be done very economically. If these measures of fortifying the flour with protein are also carried out in Turkey, there should be a very great improvement in the general health of the population, particularly of the children. Improved nutrition would aid the recovery of the thousands of children who are already affected by toxic porphyria, and improve the general health of the population as a whole. These measures could be undertaken with the assistance of the Turkish Government and perhaps help from the United States or the United Nations.

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AN UNUSUAL CYST OF THE MESENTERY

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Mesenteric cysts and enteric or enterogenous cysts are described with certain essential and characteristic differences, and their differentiation is often important in the management of a case at operation.

I should like to describe a case which does not conform to the classical and which is of sufficient rarity to warrant publication.

Incidence

Mesenteric cysts are recorded as occurring in the omentum, mesentery, and retroperitoneal regions on an average of 1 per 100,000 admissions. Beahrs *et al.*[2] report an incidence for the chylous variety of 9 per 1,000,000 admissions to the Mayo Clinic, being 9 of 174 mesenteric cysts. Gross,[1] on the other hand, apparently found a higher incidence—5 of 19 cases reported.

Description

Mesenteric cysts are described as thin-walled, usually single, lying between the leaves of the mesentery or mesocolon, or in the omentum.

As they are seldom tensely filled, they are flabby, soft tumours of a slow growth, and usually freely mobile. A dumb-bell shape is common, with the tumour bulging out on either side of the mesentery and the bowel saddling the mass.

The walls consist of connective tissue with an inner lining of endothelial cells. They may be unilocular or multilocular, and the variety known as chylangioma has been described as an anastomosing network of lymph spaces supported either by thin walls or thick septa.

The contents of these mesenteric cysts are usually serous, but may be chylous—a thick, milky, white fluid.

The absence of a muscle coat in these cysts has been emphasized by Gross,[1] but Thompson and Chambers[5] report a chylangioma in the walls of which some smooth-muscle fibres were found.

Aetiology

Various theories for their origin are propounded. Beahrs *et al.*[2] postulate that they are mainly pre-formed developmental abnormalities. He feels that a few cases may be traumatic in origin or that they are occasionally formed by degeneration of lymph nodes.

Ewing[6] states that they are all true chylangiomata due to congenital or acquired obstruction of the lacteals.

Gross[1] postulates their development from misplaced lymphatic tissues, which proliferate and accumulate fluid due to the lack of communication with the normal lymphatic system.

Lee[7] advances the theory of rupture of a lymphatic with extravasation and cyst formation, for which he has some experimental evidence.

Enterogenous cysts or duplications of the alimentary tract are all presumably embryonic aberrations, though the theory of the mechanism of origin varies. This is thought by some to be a sequestration of groups of cells, by others diverticulae of the intestinal canal, or, again, errors during vacuolation from the solid phase of development.

These cysts vary enormously in their site of development, arising anywhere from the tongue to the anus; and in their size and shape, from small cystic tumours to extensive reduplications of large segments of bowel.

Differentiation

Certain features are emphasized in the differentiation between enterogenous cysts and mesenteric cysts:

1. Mesenteric cysts are thin-walled with an endothelial lining. Enterogenous cysts are thick-walled, the walls containing muscle coats. They are lined with mucosa.

2. The duplication lies immediately adjacent to the bowel, and its musculature is so intimately associated with that of the bowel that they cannot be separated easily from one another.

3. The blood supply of an enterogenous cyst is the same as that of the adjacent bowel, so that it cannot be removed without impairing the blood supply of the adjacent intestinal segment.

The clinical picture in these two conditions may be identical in that many present with a painless, slowly enlarging mass in the abdomen, which is usually freely mobile and shows a gasless shadow on X-ray, displacing the intestines.

However, the majority will present with complications in the cyst that will alter the clinical picture accordingly.

These complications arise as result of (*a*) interference with the blood supply, (*b*) infection, (*c*) rupture of the cyst, (*d*) pressure on the bowel causing obstruction, (*e*) pressure on the contents of the pelvis, (*f*) volvulus, (*g*) intussusseption, (*h*) haemorrhage into the cyst, (*i*) accumulation of gastric acid or pepsin in the reduplications, and (*j*) obstruction of the renal tracts.

The case which I have to report presents an atypical picture and does not truly conform to either group.

Case Report

A young lady presented with a 24-hour history of pain in her upper abdomen on her 17th birthday.

She had a fairly rapid onset of symptoms which commenced in the central abdomen and were later localized in the left hypochondrium.

The pain was constant and aching in character, and was not associated with any nausea or vomiting although she was now anorexic. There was no diarrhoea or constipation.

Movement aggravated the pain and she had not slept that night.

There was nothing significant in her menstrual history and micturition was normal.

Besides a mild headache there were no other relevant symptoms, nor any previous history of illness apart from the usual childhood ailments.

On examination her temperature was 100°F., pulse 80, and respiration normal. There was no evidence of jaundice, anaemia, or lymphadenopathy. Her blood pressure was 120/65 mm. Hg.

Her abdomen was scaphoid, and the pain was indicated to be in the left hypochondrium.

Voluntary guarding was present, but with patience a tender mass was palpable. It was about the size of a naartjie. The lower edge was at the level of the umbilicus at the outer margin of rectus abdominis, and the upper position disappeared underneath the left costal margin.

There was slight downward movement on inspiration, but deep respiration was restricted by the pain. Lateral mobility could not be elicited owing to the tenderness of the mass.

Bowel sounds were present and normal. Resonance on percussion suggested bowel overlying the mass.

A blood examination showed a haemoglobin level of 14·2 g. per 100 ml., haematocrit 34%, white-cell count 9,300 per c.mm., neutrophils 79%, lymphocytes 18%, monocytes 3%, and eosinophils and basophils 0%. The platelets appeared normal. Serum amylase was less than 160 Somogyi units, and her urine contained no albumin, sugar or bile.

A straight X-ray of the abdomen showed a gasless shadow in the left upper abdomen, with a fairly well-defined outline lying opposite L 2 and L 3. It was also possible to distinguish the lower pole of the kidney and of the spleen apart from this shadow.

At laparotomy a rounded, tense, cystic mass was found in the mesentery of the upper jejunum with considerable oedema of the overlying and surrounding mesentery of this area. There was marked injection of the vessels of this region.

The mass was about 2½ inches in diameter.

It was not immediately adjacent to the bowel, there being a fingerbreadth of mesentery separating the bowel from the cyst.

On incising the overlying peritoneum, a plane of cleavage was readily found and the cyst shelled out.

Haemorrhage was minimal and easily controlled, and there was no evidence of any interference with the blood supply to the adjacent segment of bowel. The defect was closed.

Recovery was uneventful.

The cyst contained a milky white fluid and both the cyst and its contents were examined by pathologists who reported as follows: 'Sections taken from this cyst from the mesentery show the histological features of an enterogenous cyst. The lining of the cyst consists of subacutely inflamed granulation and xanthomatous tissue and contains an acute inflammatory exudate. Underlying this layer there are areas in the wall which consist of one or two layers of smooth muscle, arranged in some parts in a circular and longitudinal pattern. The remainder of the wall consists of a fairly loose connective tissue, infiltrated by a subacute inflammatory-cell infiltrate and histiocytes.'

A subsequent communication reads: 'We reviewed the section. No epithelial lining is present, but because of the presence of what appears to be a subacutely inflamed mucosa and a surrounding muscular coat, the features suggest intestinal origin'.

Analysis of the fluid from the cyst was reported on as: Total protein 1·8 g. per 100 ml., fat 12%, and specific gravity 1·011. The fluid had an opalescent white colour.

The cyst, therefore, had the macroscopic differentiating features of a mesenteric cyst and the microscopic features of an enterogenous cyst.

SUMMARY

Mesenteric and enterogenous cysts are briefly described with special reference to the differentiating features.

A case is described of a cyst which does not conform to either group in all its features.

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VAN DIE REDAKSIE : EDITORIAL

AGADIR

Kort voor middernag op Maandag 29 Februarie 1960, is die stad Agadir binne 'n paar oomblikke feitlik totaal vernietig deur 'n aardbewing. Die daaropvolgende geneeskundige ondervindings is nou so pas, veral in die Franse literatuur, geopenbaar.[1,2] Verskeie nuwe aspekte van 'n toestand wat slegs 20 jaar gelede die eerste keer duidelik erken is, verdien nou die aandag. In 1941 is Bywaters se meesterlike en reeds klassieke beskrywing van spiervergruising sonder oop wonde ('crush syndrome') onderwerp aan groot belangstelling; maar om lonende waarnemings te maak, is dit nodig om groot getalle van post-troumatiese nieraandoenings te bestudeer. Dit strek tot groot eer van Bywaters dat sy oorspronklike waarnemings in Londen gedurende die laaste oorlog so noukeurig bevestig is in Korea deur die Amerikaners, en nou in Agadir deur die Franse.

Uitgebreide spiervergruisende beserings as gevolg van ondergrondse verpersing is in 87 van die gevalle van Crosnier en sy kollegas[1] behandel in Casablanca; dié beserings is 62 keer gevolg deur 'n mate van nierskade. Eenvoudige mioglobinurie het 21 keer voorgekom, en in 19 pasiënte was daar die meer ernstige mioglobinuriese nefrose, gekenmerk deur totale anurie wat meer as 48 uur lank geduur het, met spontane herstel daarna. In die derde Franse groepering kom sterfgevalle eers voor in die sogenaamde kwaadaardige nefrose. Elf pasiënte wat behandel is sonder die kunsmatige nier, is almal oorlede; uit 11 pasiënte wat behandel is met die kunsmatige nier, was daar slegs 7 sterfgevalle. Dit is duidelik dat aanhoudende post-troumatiese anurie 'n baie swak prognose het.

Nadat die slagoffers in Agadir dae lank onder die grond vasgeknel was, was die beste behandeling dreinasie van die uitgesproke spieredeem deur spleetformasie in fascia-omhulsels, chirurgiese verwydering van duidelik nekrotiese weefsel, en daarna noukeurige kontrole van die bloed-kalium konsentrasie (op sy beste deur herhaalde elektrokardiografiese ondersoek), sodat gevalle vir die kunsnier vroegtydig erken kon word. Kalium-ophoping in die bloed was die grootste bedreiging van die Agadir-oorlewendes. Normaalweg is daar slegs sowat 3 gram kalium in die ekstra-sellulêre vog, terwyl daar intrasellulêr 147 gram aanwesig is in die gemiddelde volwassene, en die klem het deesdae verskuif van die hemoglobinurie of mioglobinurie af tot die verlies van kalium uit die selle en die daaropvolgende miokardiale prikkelbaarheid en ineenstorting.

Inwoners van Johannesburg voel elke week of twee 'n aardbewing. Baie dokters in hierdie stad het ten tyde van die Agadir treurspel gewonder wat sou gebeur as 'n stad soos Johannesburg deur 'n ernstige aardbewing getref sou word.

In Agadir is na 3 dae van reddingswerk die bevel uitgevoer om alle reddingswerk te vervang deur ontsmettingswerk. Daar was nog groot getalle lewendes vasgevang in die ruïenes, maar dit was baie warm, en die stank van die dooies en die oop rioel-stelsels was afskrikwekkend.[3] Volgens geneeskundige raadgewing was hierdie beslissing om verdere reddingswerk te staak sodat doeltreffende ontsmetting deur Duitse leërmanskappe ingestel kon word, absoluut noodsaaklik om grootskaalse epidemies te verhoed. Lt. Fordham, 'n brandweer-offisier, wonder tereg of ons westerlinge so doeltreffend sal werk aan 'n onvoorsiene natuurramp. Die eenheid en lojaliteit wat geskep is deur Kroonprins Moulay Hassan is nie maklik te voorsien in Engeland of Suid-Afrika nie, soos dit ook die geval is met die spontane wêreldwye hulp in Agadir, veral deur Franse, Italiaanse, Duitse, en Amerikaanse soldate, insluitende hul geneeskundige eenhede. Ontstellende gedagtes van hierdie aard word ondersteun deur die pleidooi van Prof. Ian Aird wat glo dat hulp in 'n nasionale natuurramp beperk behoort te word tot burgerlike persone wat verbonde is aan die Internasionale Rooikruis-organisasie, en geen ander hoegenaamd nie.[4] Gelukkig is daar ten minste die spesiale komitee van die Internasionale Federasie van Chirurgiese Kolleges en Akademies wat tans hierdie probleem van spoedige hulp ten tyde van nasionale rampe bestudeer.

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THE PYLORIC ANTRUM

It was Edkins[1] who in 1906 first postulated that the antrum of the stomach might elaborate a hormone which mediates the gastric phase of gastric secretion. He was impressed with the distinctive histological structure of the pyloric antrum, and as a result of various experiments (including the intravenous injection of extracts and the placing of food in the antrum), he considered there might be a hormonal mechanism involved. He coined the word gastrin to indicate the antral hormone.

Subsequent work cast doubt on this hypothesis. Conflicting results were obtained in animal investigations by different workers. It became apparent that histamine was present in all the extracts used, but in recent times histamine-free extracts with definite gastric secretory activity have been prepared. Studies in dogs have shown[2] that resection of the gastric antrum causes a marked decrease in the secretion of acid gastric juice when antrum resection has been carried out and when Pavlov and Heidenhain pouches have been made. This shows that the secretion of acid juice in the isolated pouches is in some way related to the presence of an antrum. Further experiments showed that, with complete interruption of the extrinsic nerve supply to the antrum and separation of vascular and neuromuscular connections to the rest of the stomach, the antrum stimulates the body of the stomach to

secrete acid gastric juice when it (the antrum) is exposed to the contents of the duodenum. Edkins' original hypothesis was in this way confirmed.

Marked increase in gastric secretion was observed after transplantation of the antrum into the colon. With this technique it was demonstrated that chronic peptic ulcers can be induced in dogs. This strengthens the view that excessive acid secretion, induced by a normal physiological stimulus (gastrin), will cause peptic ulcer.

There is also the hypothesis that acid plays an important regulatory rôle in inhibiting the hormonal mechanism of the antrum. In some way or other acid-secreting tissue inhibits the mechanism. Other physiological processes may also be involved, and although many different approaches have been made to this problem, the complete answer is not yet available. Mechanical factors may be important in the gastrin mechanism.[3] Stimulation of the isolated antrum with an inflated balloon and perfusion with an alkaline solution will cause prompt secretion of highly acid gastric juice;[4] acid will block the mechanical stimulus. Unlike a chemical stimulus, mechanical stimulation produces a constant increase in motility of the antrum. Small doses of an anticholinergic drug block the increase in motility and also the release of gastrin. Incidentally, the cytological origin of gastrin is not known. The intramural nervous tissue of the antrum would appear to be important in the endocrine mechanism.

Much work has been done to elucidate the inhibitory effect of acid on the production of gastrin. There would appear to be some local chemical interference with the formation or release of gastrin. There are studies which suggest that an antisecretory hormone may be produced by the antrum. The evidence for this has been both indirect[5] and direct.[6] Other evidence is against the concept that the antrum produces an antisecretory hormone.[7] The information which is available at present does not provide a clear answer on whether the antrum does produce an antisecretory hormone.

In summary, the pyloric antrum functions as an endocrine organ. It secretes no hydrochloric acid, but it produces gastrin which mediates the gastric or antral phase of gastric secretion. The exact nature of the chemical stimulus which releases gastrin is not known. Mechanical stimulation also releases gastrin. The formation of gastrin is inhibited by acid, thus establishing a homeostatic mechanism which terminates the gastric phase of gastric secretion. Acid also inhibits other gastric secretory stimuli. The cell origin of gastrin is unknown. Intramural nerve plexuses appear to play a rôle in the release of gastrin. Fuller details of this important physiological mechanism, which has practical implications, are given by Woodward and Dragstedt.[8]

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TAALRUBRIEK

Die Taalkomitee van die Fakulteit van Geneeskunde van die Universiteit van Stellenbosch stel voor om te gebruik:

Eng. *cachexia:*	Afr. *kakeksie*
Eng. *cachinnation:*	Afr. *kakinnasie*
Eng. *cacidrosis:*	Afr. *kakidrose*
Eng. *cacochylia:*	Afr. *kakochilie*
Eng. *cacodyl:*	Afr. *kakodiel*
Eng. *cacodylate:*	Afr. *kakodilaat*
Eng. *cacodylic acid:*	Afr. *kakodielsuur*

Maar die Komitee wil baie graag hoor hoe die lesers dink, en noem daarom enkele van die oorweginge i.v.m. die bostaande vorme.

Die Komitee sou oral vorme verkies met minder *k's*, maar dit kan nie gebeur sonder om erg willekeurig te werk nie. *Kakeksie* kom *via* Latyn *cachexia* uit Grieks *kakos* en Grieks *hexis*. Ons sou dus kakheksie kon kry, maar die *h* is in Grieks al weg en daar sou geen regverdiging voor wees om dit nou in Afrikaans te herstel nie. Maar daaruit, en uit die duidelike, oorheersende klankwaarde van die tweede *k*, volg dat ons ook nouliks daarvan *kaheksie* kan maak. Dit lyk dus of ons nie kan vrykom van *kakeksie* nie.

By *cachinnation*, van Latyn *cachinnatio*, sou 'n mens aan Latyn *machina* en Afrikaans *masjien* kon dink en dus *kasjinnasie* kon kry, maar *sj* binne-in 'n woord is vir Afrikaanssprekendes 'n vreemde (rige) klank en dus minder aanneemlik. Die Komitee meen dus ons moet maar *kakinnasie* sê en skrywe.

Cacidrosis kom van Grieks *kakos* en *hidros*. 'n Mens sou op grond van 'n letteruitspraak *kasidrose* kon oorweeg, maar dit sou dan beteken dat Grieks *kakos* in sommige gevalle as *kak-* en in ander gevalle as *kas-* weergegee sou word, wat darem nie deug nie. Daarom sou *kaksidrose* ewe min deug. Ons meen dus dat daar vir *kakidrose* die meeste te sê is.

Doen ons dit, volg daaruit dat ons met *cacochylia* weinig keuse meer het: ons is vanself aangewese op *kakochilie*, en met Engels cacodyl(*ate/-ic*) op *kakodilaat* en *kakodiel*.

Dit was nie vir die Komitee binne enkele minute duidelik dat dit die mees verdedigbare vorme is nie, en lesers kan dus verskil. As u dus ander gemotiveerde beskouinge hieroor het, laat asseblief van u hoor.

CONGENITAL CERVICAL CYSTS AND FISTULAE*

A. LEE MCGREGOR, *Johannesburg*

Congenital cysts and fistulae of the neck are reported in sizable numbers from large surgical centres. Most surgeons and general practitioners see such cases but rarely.

* An invitation lecture delivered at the Surgical Forum of Prof. D. J. du Plessis at the Medical School, Johannesburg.

The object of this paper is to present a personal series of 48 cases seen in private practice between 1941 and 1960.

Material: Branchial maldevelopments 21, thyroglossal anomalies 27, total 48. Branchial or visceral arch maldevelopments comprised:

(*A*) Pre-auricular fistulae, 4 cases, all of which required operation; and

(*B*) branchial anomalies. These include:

(*a*) Branchial fistulae, 1st cleft, 2 cases of which 1 was operated on; and 2nd cleft, 8 cases, in which the fistula was symmetrical and bilateral in 3. Thus 11 fistulae required operation. Three cases were not operated on.

(*b*) Branchial cysts, 4 cases were operated on.

Aetiology

A. *Pre-auricular sinuses* are present at birth. They may become fistulae if infection of the track occurs and an abscess bursts. They are not actually of branchial origin, but result from failure of perfect coalescence between 2 of the 6 tubercles which develop around the posterior end of the first branchial cleft and form the pinna or auricle (Fig. 1). Tubercle 1 forms the tragus, 2 the crus helicis, 3 the helix, 4 the anti-helix, 5 the anti-tragus, and 6 the lobule.

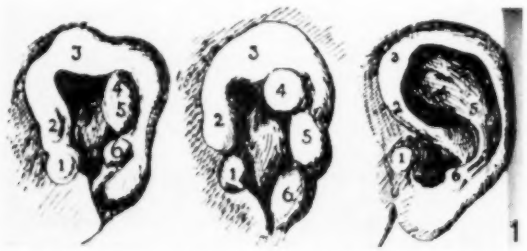


Fig. 1. Development of auricle. The six tubercles are shown. The usual type of pre-auricular fistula is due to a faulty fusion of tubercles 1 and 2. (Re-drawn from Prentiss and Dreg.) From 'Synopsis of surgical anatomy' by courtesy of John Wright and Sons, Bristol.

This anomaly almost always affects the union between tubercles 1 and 2 and thus opens between the tragus and crus helicis or on this crus (Fig. 2).

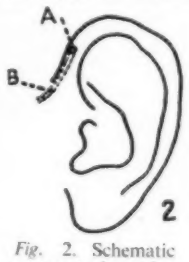


Fig. 2. Schematic representation of a pre-auricular fistula: A = fistula, B = track of fistula. From 'Synopsis of surgical anatomy' by courtesy of John Wright and Sons, Bristol.

The 4 patients in the series comprised 2 females aged 8 and 20, and 2 males aged 18 and 43.

In both females there had been a series of abscesses over a period of 3 years. In the males symptoms had existed for a few months only.

Clinically these sinuses are usually very small. They resemble a tiny pigmented spot. They are frequently unobtrusive and overlooked. I once visited the home of a colleague whom I had not seen for 15 years. He questioned the value of describing rarities like pre-auricular fistulae about which I had written and which the general practitioner never sees. Just then the doctor's son, aged 12, entered the room and his pre-auricular fistula was obvious.

Such sinuses have an hereditary tendency and illustrate the law that congenital anomalies are often multiple. The older of the two girls reported here has a sister, aged 23, with bilateral pre-auricular fistulae which have never caused trouble.

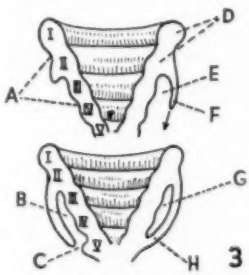
There may be associated cleft palate or harelip, and one of the cases in this series was associated with a thyroglossal cyst. In most cases the sinus causes no trouble and the possessor may be unaware of it. The sinus is lined by stratified squamous epithelium. The track passes downwards and forwards. When infection supervenes the condition presents as a fistula, which opens in front of the tragus, or there may be a red swelling due to an abscess. It is exactly in the position of the pre-auricular gland for which it may be mistaken. The abscess bursts, heals, bursts again, and so on.

Treatment is only necessary if the track is infected. It entails excision of the track and the abscess cavity. The lower part of the excised track may be slow in healing because of the presence of infection.

This operation is not a very satisfactory procedure. Excision cannot be deep over the parotid because of the local anatomy. Cells are displaced by the pathological changes so that late recurrence is common. Two of the cases now reported have recurred within a year of operation.

The writer has seen no instance illustrating the statement that the fistulous track may take a complicated course traversing the parotid and opening in the neck. These are probably instances of the much rarer condition of fistula of the first visceral cleft.

B. *Branchial anomalies.* The developmental origin of branchial fistulae and cysts has been the subject of much discussion, and final agreement has not yet been reached (Fig. 3). Briefly, the neck and pharynx are formed from 5



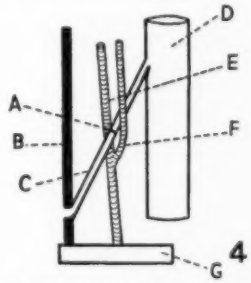


Fig. 3. Figure illustrating method of formation of a branchial cyst and branchial fistula; A = visceral clefts, B = cervical sinus, C = failure of fusion of second arch, causing branchial fistula. D = visceral arches, E = cervical sinus, F = downgrowth of second arch, G = branchial cyst, H = fusion of second arch with skin of neck. From 'Synopsis of surgical anatomy' by courtesy of John Wright and Sons, Bristol.

Fig. 4. Course of a branchial fistula; A = cleft membrane, B = skin, C = fistulous track, D = pharynx, E = internal carotid, F = external carotid.

branchial or visceral arches, as Frazer preferred to call them. Each arch has a muscle mass, a plate of cartilage, a nerve, and an artery. Between the bars are depressions—the branchial clefts externally, lined by squamous epithelium, and the branchial pouches internally, lined by ciliated columnar epithelium. They are separated by the cleft membrane. Developmentally there is never a complete canal from without inwards. If the cleft membrane is absent it is because it is destroyed, e.g. by inflammation or by surgical interference. According to His the second arch grows more

rapidly than successive arches, and comes to overhang them. Thus the pre-cervical sinus is formed. Ultimately it fuses with the fifth. Should fusion be imperfect, a branchial fistula results. Normally the pre-cervical sinus becomes first a buried space and then disappears entirely. If the space persists, a branchial cyst results.

In 1926 Frazer produced evidence to show that the pre-cervical sinus was obliterated from its depths and not by the overhanging of the second arch. During this process a groove exists into the top end of which the second branchial cleft opens, the third and fourth clefts opening into the lower end. Thus, according to Frazer, the situation of the external opening of a branchial fistula at birth, at the anterior border of the sternomastoid, gives no indication of its cleft origin.

The course of the fistula. From its opening just above the sternoclavicular joint at the anterior border of the sternomastoid, the fistulous track passes subcutaneously to the level of the upper border of the thyroid cartilage, where it pierces the deep fascia. It then dips beneath the posterior belly of the digastric and stylohyoid, crosses the hypoglossal nerve and the internal jugular vein, and traverses the fork formed by the bifurcation of the common carotid, the internal carotid being behind and the external in front. The track then lies on the middle constrictor of the pharynx, crossing the glossopharyngeal nerve and the stylopharyngeus muscle, to pierce the superior constrictor and terminate on the posterior pillar of the fauces behind the tonsil (Fig. 4). It does not end in the intra-tonsillar cleft (supra-tonsillar fossa).

Wilson[1] states that the fistulous track receives an investment of muscle from the platysma below and the stylopharyngeus above. If the latter is well developed the external orifice of the fistula will pucker on swallowing. This was demonstrable in 2 patients in this series.

The track is lined by squamous epithelium posterior to the cleft membrane, and by ciliated columnar epithelium in its anterior entodermal portion.

Anomalies of the second cleft may occur as (so-called) complete fistulae, or portions only of the track may persist. Thus there occur sinuses with an external opening and extending inwards only for short distances. If the deeper part only persists, a cyst results. It is probable that entodermal pouches, opening into some part of the pharynx, are more common than is currently thought. If they accumulate secretions or foodstuff, they may cause symptoms such as the periodic discharge of purulent material. Doubtless some are not suspected.

Clinical Features

Fistulae are bilateral and symmetrical in about a third of the cases. There is both a hereditary and a familial tendency. A brother and sister are included in this series. In another case the mother of a girl of 10 with bilateral fistulae, had herself had an opening above the sternoclavicular joint since birth. Exploration showed that the track was only an inch long. A similar case occurs in this series in a boy aged 5 years.

The complaint is of an annoying, clear, or yellowish discharge. Occasionally a swelling forms just proximal to the opening, which might show a mild local inflammation that subsides when the collection is discharged. The external opening in all the cases here reported was extremely small—about the size of a pin head. It is different in colour to the surrounding skin and within an inch of the sternoclavicular joint near the lower end of one or both of the 'bonnet strings' formed by the sternomastoid muscles.

This series comprises 11 patients with second cleft fistulae. Seven were males and 4 females. The ages varied from 3 to 22 years, the average age being 10·9 years. Three of the cases were bilateral and symmetrical, the external openings being tiny and just above the sterno-clavicular joint. Three patients, all females, refused operation.

Surgery

The only treatment is total extirpation. Several practices are referred to merely to be condemned: Nothing should be injected into the fistula—neither sclerosing solutions as a method of treatment, nor contrast media for radiography. Sclerosing solutions are worthless and the use of contrast media unnecessary, and both may convert a clean track into an infected one. These tracks are surprisingly thick-walled structures, with considerable tensile strength, which can be followed throughout their course by the surgeon.

The injection of methylene blue to delineate the track merely confuses the surgeon, because before long, not only the track, but also all the surrounding tissues share in the cerulean obscurity. The sooner an operation is performed, once teething is complete (i.e. after 2), the better. This avoids difficulties which might be caused by obscuration of fascial planes due to attacks of lymphadenitis, etc.

The exposure is by a 2-tier incision in the line of the neck creases, as pointed out by Hamilton Bailey. The lower incision enables the fistulous opening to be mobilized and the track to be exposed (where it is freed from platysma and subcutaneous fat) up to the level of the upper border of the thyroid cartilage, by retracting the top flap. The second incision is at the level of the hyoid bone. It is deepened through deep fascia and the posterior belly of the digastric is exposed. The track is now brought into this incision by freeing it from the lower flap. It may be crossed by the common facial vein which is secured between ligatures. By retraction of the digastric, the carotids are exposed as is the hypoglossal nerve.

It is unnecessary to do a great deal of dissection because the relationship of the track to the structures mentioned is not intimate. The track is a strong tube when the fistula is complete. When the sinus is short the vestige is much more tenuous. The anaesthetist pushes the tonsil outwards with his forefinger, while the surgeon makes gentle sustained traction on the vestige, pushing surrounding structures off the track with the handle of the knife. The side of the pharynx comes to meet him, so to speak. Using this technique, it is then possible to transfix the track at the superior constrictor and stylopharyngeus with a thread of catgut on a 'tickey'-sized needle and to tie it off and remove it. In one case the track came away at its pharyngeal attachment before experience of the manoeuvre had been acquired. This was of no consequence in the convalescence of the patient.

The deep fascia is sutured in the upper incision with catgut. No drainage is used. Clips are removed in 48 hours, and the patient goes home within the week. If the fistula is bilateral the second side is done in the ensuing school holiday. There have been no complications (Fig. 5). These cases have been

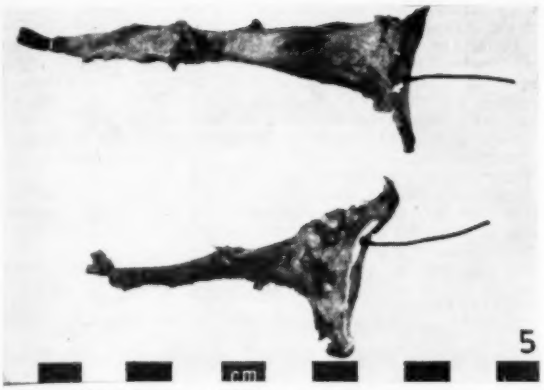


Fig. 5. Bilateral second cleft fistulous tracks.

treated over a period of 17 years, 2 of them within the last year. If the fistula has been removed *in toto* the patient is cured. The transverse scars are usually inconspicuous.

Branchial Cysts

In large series of branchial remnants, such as those quoted later, branchial cysts are found much more commonly than fistulae—i.e. in the proportion of 3 : 1.

In this small series there were 4 cysts. The diagnosis can only be verified by the operative findings and histology. The cyst is due to persistence of some buried part of a branchial cleft or visceral pouch. There is no communication with or attachment to skin. Cases have been described where cysts of entodermal branchial origin have been in communication with the pharynx, the opening having become shut off. Cysts of ectodermal origin (vastly commoner) are lined by squamous epithelium, while entodermal cysts have a columnar epithelial lining. The 4 cases reported here belonged to the former and commoner group. Lymphoid tissue is found in the wall of the cyst. The contents are clear and contain cholesterin crystals which may impart a shimmer to the fluid.

The great majority of these cysts are of second branchial-cleft origin. If the cyst extends inwards it will lie between the carotids. If it lies posterior to internal and/or common carotid it takes origin from the third cleft, since the internal carotid, being the artery of the third branchial arch, lies anterior to the 3rd cleft. One such case occurred in this series. This was a man aged 54 who presented in 1946 with a coconut-sized mass under the upper left sternomastoid. He had known of a small mass there for over 20 years. A year previously it had begun to enlarge rapidly. The mass was elastic in consistency and thought to be a degenerating neoplasm. At operation the tumour was found to extend up to the base of the skull between the arch of the atlas and behind the great vessels. During the removal the vagus nerve was found to be densely adherent to the mass. Convalescence was uneventful. Histologically it was found to be a branchial cyst.

Unlike fistulae, branchial cysts usually appear in adult life, often in the third decade. Bilaterality is rare.

Clinically the diagnosis is presumptive, since breaking-down glands or tuberculous abscesses are much more common. The cyst is under the sternomastoid, about its middle and often behind the angle of the jaw in the area of the tonsillar gland. The mass may feel cystic or solid. Sometimes a helpful sign is that the mass feels half empty. It usually lies in front of the carotid sheath, but may extend deeply. It may become infected *via* the lymphoid tissue in its wall, and present as an abscess which requires drainage in the first instance with later excision. The age range is 9 - 54. The average age of the 4 patients in the series was 40·5 years, there being 2 of each sex. In one case the cyst was densely adherent to the vagus nerve though no cough was caused thereby as has been reported.

Surgically excision may be very difficult because of deep extension and adhesions to neighbouring structures. This is especially the case if infection has occurred. It is well to remember that many cysts develop layers of fibrous tissue around them by pressure on neighbouring tissues during slow expansion. A plane can be found within this false capsule out of which the cyst may be shelled.

The 4 patients now recorded had no postoperative troubles and 3 are well after periods of 5 or more years.

The patient mentioned above, where the vagus was dissected off the cyst wall, has remained well since 1946. He recently presented with the suggestion of an indefinable softness beneath the scar which may be the herald of recurrence.

Some branchial cysts may therefore present much more intricate surgical problems than fistulae.

First Cleft Anomalies

The ectodermal portion of the first cleft takes part in the formation of the external auditory meatus. The corresponding entodermal portion, or pharyngeal pouch, becomes the tympanic cavity of the middle ear and the Eustachian (auditory) tube. The line of obliteration of the ectodermal portion of the first cleft extends from within the external auditory meatus to just below mid-point of the mandible between it and the hyoid bone. Should the edges of the first cleft unite over it, a skin-lined tunnel will be formed (Fig. 6). It will open on the skin between the mandible and hyoid bones below. The opening is not related to the anterior border of the sternomastoid as usually stated. The track extends up and back, deep to or through the parotid, and superficial to the posterior belly of the digastric muscle and the external carotid (all derivatives of the second arch). The relationship of the track to the facial nerve is uncertain though it is usually described as being superficial.[2] The track stops at or may open into the external auditory canal. These auro-cervical fistulae are very rare. Neel and Pemberton,[3] from the Mayo Clinic, found no first cleft

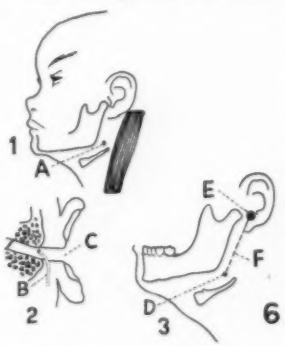


Fig. 6. 1, External opening of first branchial cleft fistula; A=sinus opening on surface. 2, Course of first cleft fistula; B=fistulous track, C=external auditory canal. 3, Coronal section showing termination of first cleft fistula in external auditory canal; D=skin opening, E=external auditory canal showing fistulous opening, F=course of fistula. From 'Synopsis of surgical anatomy' by courtesy of John Wright and Sons, Bristol.

anomalies in 319 cases of branchial maldevelopment. Rankow and Hanford[4] recorded 3 first cleft lesions out of 160 branchial defects at the Columbia Presbyterian Hospital, New York.

Gross[5] reviewed 308 cases of branchial anomalies of which only one involved the first cleft.

Altogether about 13 such fistulae are recorded, 10 of which were in female patients, as were the 2 cases here described.

Clinically these cases behave much as cases of second cleft remnants. There is always an external opening in the submaxillary area between mandible and hyoid bone. There may be a slight discharge in the first few months of life. There may be no discharge for years. Abscess formation occurs if the submandibular opening becomes blocked. There may be a discharge from the ear. The track is lined by squamous epithelium.

Cases have been reported from 1 to 45 years old. The 2 cases reported illustrate variations of behaviour.

Case 1

Miss S.E., referred by Dr. I. Effren of Springs, was first seen in 1948 at the age of 1 year and 11 months. She was born with a 'dimple' in relation to the horn of the hyoid on the right. There had been no discharge. She was referred for consultation because latterly a swelling had occurred beneath the dimple.

On examination a tiny fistula was found. There was a suggestion that a track could be felt running back and up. The track and the dimple were very mobile. A note made at the time stated that this was not a second cleft anomaly, and observation was advised since there had been no discharge or signs of inflammation.

I was undecided as to the genesis of the condition, since auro-cervical fistulae had not been described up to that time. The child was not seen again for exactly 10 years. In 1958 her parents stated that 2 weeks previously a slight smelly discharge had occurred from the neck opening and from the ear. She was found to have a large swelling beneath the blocked fistulous opening, which was hard and tender. There was no discharge. An otologist reported that the ear was normal. The family doctor was advised to drain the abscess. The parents were told an ablation of the track would be necessary later. Four days after the consultation an opening appeared on the skin near the fistulous one and discharged pus, which also extruded from the external auditory meatus. Dr. Effren was able to pass a probe throughout the length of the track (Fig. 7). Three weeks later the fistulous track was excised.

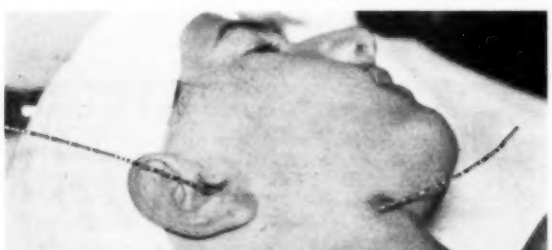


Fig. 7. A probe throughout the length of a first cleft fistulous track.

Operation (11 November 1958): A ureter catheter was placed through the fistulous track emerging at either end. The face was left exposed so that the anaesthetist could watch for muscle twitching. An incision was made extending from mastoid process, and passing an inch behind the angle of the jaw to protect the ramus colli mandibularis of the facial nerve. It then encircled the sinuses which were mobilized. The track was dissected out, passing deep to the parotid gland and over the posterior belly of the digastric muscle. Attention was given to exact haemostasis, and all tissue to be divided was stimulated by compression with dissecting forceps while the face was observed. No twitching was seen at any time. The track entered the auditory canal at the junction of cartilage with bone and was excised. The facial nerve was not seen. No drains were used. The opening in the auditory canal was not closed. Following the operation there was a complete facial paralysis.

Convalescence was otherwise uneventful.

Electrical reactions, carried out by Drs. Adler and Hoffman soon after the operation, indicated that the prognosis should be good following on a prolonged course of physiotherapy. Some degree of recovery took place slowly.

At this time, 18 months after operation, Dr. Effren tells me that the face is symmetrical in repose, but considerable deformity occurs on laughing.

There is no epiphora and the eye can be almost completely closed.

This is the first recorded case where facial paralysis has followed excision of the fistulous track. The second case behaved differently.

Case 2

S.W., female, aged 1 year and 4 months, was first seen in 1952. She was born with a 'hole' in the left submandibular region just above the great horn of the hyoid. Rarely there extruded a thread of thick yellow material. At the age of 10 months a big abscess formed which was lanced with extrusion of pus. This operation was repeated twice in the ensuing 3 months. The discharge has continued. For the rest the child was well, except for a query in the notes as to whether the mouth was asymmetrical.

On examination there was a large scab in the left submandibular area with a considerable amount of pus beneath it. The case note at the time states that the sinus was not of second branchial origin, but could be related to the first cleft.

Under anaesthesia the pus was released and a probe passed for 1¼ inches towards the external auditory canal. No sinus was found in the canal.

The child did not return for excision of the sinus and efforts to trace the family have failed.

This was a first cleft sinus which required excision when infection had subsided.

There is now sufficient knowledge of first cleft sinuses or fistulae to recommend their excision since they will at some time give rise to symptoms, such as discharge from the neck or the ear, or abscess formation. The patient or parents should be warned about the possibility of damage to the facial nerve.

THYROGLOSSAL CYSTS AND FISTULAE

Twenty-seven cases of thyroglossal cysts or fistulae were seen from 1942 to 1955. The features and treatment of these conditions are briefly reviewed here. Eleven of these had been operated on for excision of a cyst or fistula previously.

Unlike that of branchial anomalies, the aetiology is well known. From behind the tuberculum impar, the site of the future foramen caecum, at the junction of the anterior two-thirds with the posterior third of the tongue, a solid rod of cells, which later becomes canalized, passes down in the midline between the genioglossi to the upper border of the thyroid cartilage. There it is slightly deflected right or left by the keel of this cartilage. It forms the thyroid gland. The pyramidal lobe is a remnant of the duct. The upper part normally disappears. The development of the body of the hyoid bone results in a deflection of the duct posterior to the bone.

The surgical cure of cysts or fistulae of the duct depends, among other factors, on removal of the central part of the bone, since the remains of the duct cannot be completely removed otherwise because of their close inter-relationship (Fig. 8). As the development indicates, a thyroglossal fistula

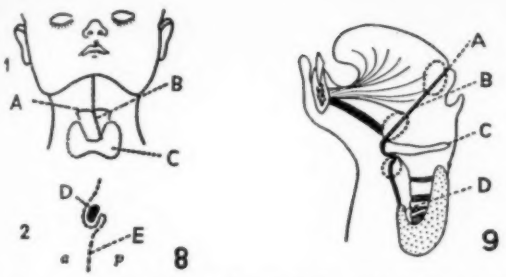


Fig. 8. 1, Course of thyroglossal duct—anterior view; note divergence to left below upper border of thyroid cartilage; A=upper border of thyroid cartilage, B=thyroglossal duct, C=thyroid gland. 2, Lateral view showing relationship between duct and hyoid bone; D=hyoid bone, E=thyroglossal duct, a=anterior, p=posterior. From 'Synopsis of surgical anatomy' by courtesy of John Wright and Sons, Bristol.

Fig. 9. Thyroglossal duct, and sites occupied by thyroglossal cysts; A=foramen caecum, B=thyroglossal duct, C=hyoid bone, D=pyramidal lobe of thyroid gland. From 'Synopsis of surgical anatomy' by courtesy of John Wright and Sons, Bristol.

cannot be congenital in the true sense of the word. Cases reported as such must be due to rupture of a cyst *in utero*. Thyroglossal cysts may be lined by flattened or columnar epithelium. Occasionally thyroid or lymph tissue occurs in the wall. The contents may be clear with cholesterin crystals. Cysts are often infected. Cysts may be suprahyoid or infrahyoid, as the majority are. Most of these cases occurred in relation to the hyoid bone or thyroid cartilage (Fig. 9).

Clinical Features

Thyroglossal cysts may occur for the first time at any age. The ages in this series vary from under 2 years to 60. The average age was 21·8 years. Sex incidence varies. In the 27 cases here reported there were 16 males and 11 females. The length of the history varied from a few weeks to 40 years—the average being 3·1 years. Cysts vary in size and may become as large as an orange. They move with swallowing and some move up on projection of the tongue. Thyroglossal cysts are much more liable to infection than those of branchial origin. Out of 9 operations for cysts, 4 were infected. They burst or are incised, reform, or remain as a sinus. In rare cases carcinoma of the thyroid type may occur.

Treatment is surgical. The operation is performed in a non-inflammatory phase, though there may be pus in the cyst. Incision is transverse and surrounds the fistula. The cyst or fistula is dissected and traced to the hyoid bone. This structure is shaped like an inverted L. The sternohyoid and mylohyoid muscles are freed from the body of the bone, which is pulled forward with a sharp hook, and the central ½ inch is cut loose with bone forceps, the cyst or fistulous track remaining attached. The upper shelf of the hyoid extends farther back than expected, and must be removed. Opinions differ on how to proceed further. The procedure of Sistrunk, whereby a cylinder of tissue is cored out, upwards and backwards, from the hyoid to the foramen caecum (45° angle), is popular. It has not been used here, because the procedure is a difficult and blind one in so far as any upward extension of the remnant is concerned Once the central part of the hyoid with cyst (fistula) attached is freed from the rest of the bone, the muscle tissue of the geniohyoids and genioglossi is carefully separated from this bone fragment, and an upward extension of the track is looked for. It is as a rule string-like and quite different in appearance to muscle. It is grey or brown. It may widen as it approaches the tongue and be trumpet-shaped.

It is traced up between the genioglossi. The procedure is much facilitated if the anaesthetist puts his finger on the area of the foramen caecum as a guide to the final part of the dissection. It is ligated and cut off at the level of the foramen. One or two stitches approximate the muscles. The gap in the hyoid is not closed. If no upward extension is found, no further dissection is carried out. Drainage is unnecessary. Rarely a nodule which resembles a thyroglossal cyst may be the only thyroid tissue the patient possesses. The surgeon must satisfy himself that the tissue he plans to remove is in reality a cyst. If it is solid, the presence of many supplying blood vessels may give a clue to its nature. A small incision is then made over the thyroid gland itself, and its existence or otherwise is determined. If the upper nodule is the only existing thyroid tissue, it is left *in situ*.

Of the 13 cases operated on in this series, 9 were cysts and 4 sinuses. Four of these were recurrences following previous operations elsewhere. In all cases the central part of the hyoid bone was removed, and in 7 a remnant of the thyroglossal track was traced to the tongue. This is a higher percentage than reported by others.

In one case the upper end of the track reached the vallecula and not the foramen caecum. In another the track divided and 2 string-like bands were traced to the tongue.

There were no postoperative complications. The patients were treated at periods from 5 to 17 years ago. There have been no recurrences.

RÉSUMÉ

The series comprises 48 patients with branchial or thyroglossal anomalies seen in private practice. Eleven had had previous operations designed to cure the conditions. Of the total, 30, comprising 33 operations, elected surgery. Operations were performed for 4 pre-auricular fistulae, one fistula of the first cleft, 11 fistulae of the second cleft, 4 cysts of the second or lower clefts, and 13 thyroglossal cysts or fistulae.

RESULTS

Two pre-auricular fistulae were cured and two were not cured. The first cleft fistula was cured, but the patient was left with a partial facial paralysis.

The 11 second cleft fistulae were cured.

One branchial cyst shows signs (8 years after operation) of what may be a recurrence.

None of the thyroglossal cysts or fistulae have recurred within 5 to 17 years after operation.

DISCUSSION

Comparison of branchial with thyroglossal remnants dealt with in this series discloses the following:

Aetiology. Not settled regarding branchial anomalies, whereas the origin of thyroglossal anomalies is known.

Situation. Branchial—lateral, thyroglossal—medial.

Presentation. Primary fistulae include most branchial cases. Thyroglossal fistulae are secondary. Branchial fistulae

are present at birth. Thyroglossal fistulae cannot be (developmentally) congenital. Branchial cysts occur at a variable time after birth. Thyroglossal cysts may exist at birth or appear at any age.

Multiplicity. Branchial fistulae are bilateral in 30% of cases or more. Thyroglossal anomalies are always single.

Previous operations. None in branchial anomalies, common in thyroglossal operations.

Totality. Branchial fistulae are complete but for rare exceptions. Thyroglossal remnants stop at the hyoid bone in most cases.

Relationship to important vessels and nerves. Intimate in branchial anomalies, non-existent in thyroglossal anomalies.

Nature of operation. Entirely satisfactory in branchial fistulae. Less satisfactory in thyroglossal fistulae.

Malignancy. In branchial cysts rare and doubtful; in thyroglossal remnants exceedingly rare.

SUMMARY

1. Branchial and thyroglossal cysts and fistulae are curable conditions providing the surgeon is familiar with their developmental anatomy.

2. Auro-cervical or first cleft fistula is an established entity.

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SERUM HAPTOGLOBINS IN AFRICA*

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The haptoglobins are serum proteins that combine with haemoglobins. They are part of the α_2-glycoprotein fraction and were first detected by Polonovski and Jayle in 1938.[1] Peroxidase activity is developed when haemoglobin forms a complex with haptoglobin,[2] and this property has been used to measure the concentration (or index) of haptoglobin in normal and abnormal sera.[3] The haptoglobin concentration has been found to be raised in a variety of diseases in which there is inflammation and tissue necrosis, including acute pyogenic infections, rheumatic fever, and some forms of cancer,[4] as well as in patients with rheumatoid arthritis.[5]

By means of starch-gel electrophoresis, Smithies[6] identified 4 different haemoglobin-binding proteins, which are the same as haptoglobins. Smithies and Walker[7] demonstrated that European populations have 3 major patterns, termed haptoglobin types 1 - 1, 2 - 1, and 2 - 2. These phenotypes are under the control of a single pair of allelomorphic genes designated Hp^1 and Hp^2. [8,9] Genotype Hp^1/Hp^1 produces haptoglobin type 1 - 1 (Hp1 - 1); Hp^2/Hp^1 produces Hp2 - 1; and Hp^2/Hp^2 produces Hp2 - 2.

Until quite recently, publications[10-21] concerning the haptoglobins emphasized that the Hp^2 gene is commoner than the Hp^1 in all European populations studied, and that in Africa Hp^1 is much more frequent than Hp^2. The rather simple story of a single pair of allelomorphic genes producing 3 genotypes in man, and the co-existence of these haptoglobins types in several populations providing a true state of polymorphism, became more intricate with the publication by Allison *et al.*[13] of an apparent fourth group, in which no haptoglobin at all is present, which they designated Hp0 - 0. Since then further rare modifications have been described. Furthermore, Sutton *et al.*[14] mention the presence of 5 different haptoglobins in the type 2 - 2 individuals and 6 haptoglobins in the 2 - 1 heterozygotes. The apparently clearcut racial distinction between Europeans and West African Negroes has also become more complex and less clear with publications on the incidence of the haptoglobins in Swedish Lapps;[15] in Japanese populations;[16] and in Malays, Chinese, and Indians in Malaya.[17]

TABLE I. DISTRIBUTION OF HAPTOGLOBIN TYPES IN VARIOUS POPULATIONS

	No.	*Gene frequency*		*HpO*	*References*
		Hp^1	Hp^2		
Europe					
Basques (Spain)	107	0·39	0·61	0·01	13
British	218	0·38	0·62	0·03	13
British	114	0·42	0·58	—	10
Swedish Lapps	329	0·28	0·72	0·02	15
Finns	891	0·36	0·64	0·002	35
Danes	1,033	0·40	0·60	—	9
Norwegians	1,000	0·36	0·64	—	36
Swedes	1,003	0·37	0·63	0·03	37
Italians	466	0·38	0·62	0·01	10
French	406	0·40	0·60	—	38
Americas					
Alaskan Eskimoes	418	0·29	0·71	—	12
Anaktuviak Eskimoes	57	0·50	0·50	0·04	12
Athabascan Indians	284	0·42	0·58	0·01	12
Apache Indians	98	0·59	0·41	—	14
US Whites	68	0·43	0·57	—	14
US Whites	54	0·38	0·62	—	14
Canadian Whites	49	0·44	0·56	—	5
US Negroes	48	0·59	0·41	0·10	14
US Negroes	760	0·54	0·46	0·04	39
Venezuelans (Caracas)	208	0·55	0·45	—	40
Central American Non-Maya	170	0·57	0·43	0·01	41
Central American Maya (Less Lacandon)	414	0·59	0·41	—	41
Central American Lacandon	31	0·93	0·07	0·10	41
Peruvian Indians	173	0·73	0·27	—	48
Africa					
Yoruba (Nigeria)	99	0·87	0·13	0·32	13
Yoruba (Nigeria)	30	0·72	0·18	0·23	10
Habe (Nigeria)	120	0·60	0·40	0·27	46
Fulani (Nigeria)	111	0·76	0·24	0·37	46

* Read in modified form at the Conference on Biochemical Anthropology held at Yellow Springs, Ohio, United States of America, in July 1960.

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Liberia and Ivory Coast Negroes	142	0·70	0·30	—	21
Liberia and Ivory Coast Negroes	614	0·72	0·28	—	14
Gambia	157	0·70	0·30	0·40	11
Ibo	70	0·49	0·51	0·48	11
Metropolitan Congo	186	0·60	0·40	0·05	42
Non-metropolitan Congo	468	0·57	0·43	0·21	42
Pygmy (Congo)	125	0·40	0·60	0·31	42
Zulus (South Africa)	113	0·53	0·47	0·03	18
Bushmen (South Africa)	113	0·29	0·71	0·02	18
Hottentots (South Africa)	59	0·51	0·49	—	18
Cape Coloured (South Africa)	88	0·47	0·53	—	18
Xhosa and Msutu (South Africa)	315	0·55	0·45	0·05	43
Asia and Oceania					
Malaya-Malays	236	0·24	0·76	0·01	17
Malaya-Chinese	167	0·28	0·72	0·01	17
Malaya-Indians	219	0·09	0·91	0·02	17
Asiatic Indians	74	0·18	0·82	—	14
Japanese (Sapporo)	349	0·24	0·76	0·01	16
Micronesians (Marshall Islands)	52	0·58	0·42	—	47
Borneo	22	0·50	0·50	—	11
Tongans	200	0·60	0·40	—	44
Australian Aborigines	133	0·17	0·83	0·01	45

Before discussing the current trends in the interpretation of and the difficulties in relation to the thus far very limited and discrepant African series, some information will be presented on the haptoglobin distribution in Bushmen, Hottentots, Cape Coloureds and Zulus, based on the field work of Dr. J. S. Weiner and myself, and published in collaboration with Drs. N. A. Barnicot and J. P. Garlick in *Nature* in December 1959.[18]

Blood samples were collected from (1) Naron Bushmen living their traditional hunting and food-collecting life and from Bushmen employed on farms near Khanzi in Bechuanaland; (2) Nama-speaking Hottentots or Khoi-Khoi in the Richtersveld region of Namaqualand; (3) Cape Coloureds in the Springbok and Steinkopf areas of Namaqualand, whose gene pool consists essentially of Caucasoid (Dutch, German and English) and Hottentot components with possibly slight Bushman and minimal, if any, Negroid admixture; and (4) Zulus working in Johannesburg.

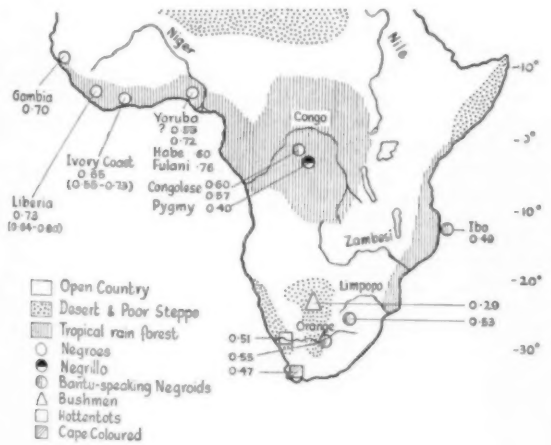

Fig. 1. Map of Africa: Frequency of Allele *Hp*.[1]

The techniques used on the sera by Drs. Barnicot and Garlick were essentially those outlined by Smithies[6,19] and by Poulik.[20]

The most striking feature is the low frequency of the haptoglobin Hp^1 in the Bushmen as compared with the other 3 South African series, which are closely similar to one another in this respect. The Hp^1 frequency in the Zulus, Cape Coloured and Hottentots is lower than the lowest limit of the range reported for Negroes from Liberia and the Ivory Coast and the Nigerian Yoruba.[13,14,21] No haptoglobins were located in 4 Bushmen and 3 Zulus. These were excluded in the calculation of the gene frequencies since the genetical significance of this phenotype, which thus far is very common only among the Yoruba and some Liberians, is still not clear. Some typical examples of the modified 2 - 1 phenotype (2 - 1M) were found in the Zulu sera, but not in the other populations. However, in the Bushmen and Coloureds a few sera were noted in which the slower $\alpha\beta$ bands were relatively weak although the Hp^1 band was somewhat weaker than the first $\alpha\beta$ band.

TABLE II. THE INCIDENCE AND GENE FREQUENCY OF HAPTOGLOBIN PHENOTYPES IN SOME SOUTH AFRICAN PEOPLES

		Haptoglobin phenotypes					*Gene frequency*	
	No.	*1-1*	*2-1*	*2-1M*	*2-2*	*0-0*	Hp^1	Hp^2
Bushmen:								
(*a*) Tribal	71	7	29	0	34	1		
(*b*) Farms	42	5	11	0	25	1		
Total	113	12	40	0	59	2	0·29	0·71
Hottentots:								
(*a*) Random	34	10	16	0	8	0		
(*b*) Related	25	8	9	0	8	0		
Total	59	18	25	0	16	0	0·51	0·49*
Cape Coloured	88	17	49	0	22	0	0·47	0·53
Zulus	113	36	45	3	29	3	0·53	0·47

* Standard error=0·05

Information concerning the distribution of serum-protein variants in Africa is still very limited, and our material represents only a fraction of the widely dispersed Bantu, Bushman, and Hottentotoid peoples. Consequently, only tentative opinions rather than conclusive assessments are feasible.

Bushmen are distinct from other African populations both in morphology and blood-group frequencies,[22,23] and it appears that their haptoglobin frequencies may also be unusual. It is interesting that the general serum-protein pattern of the Okavango Bushman and Bantu tribes, as determined during the University of Cape Town 1952 Expedition, is quite distinct from the 'normal' pattern for urbanized Bantu groups in Cape Town.[24] Furthermore, the so-called 'abnormal' pattern of the Bushmen (which is probably normal for the Okavango and adjacent Kalahari regions), is related directly to the distance of their usual habitat from the Okavango River and its associated malaria and bilharzia. From this point of view it seems that there is an advantage in hunting and food-collecting in the desert away from the rivers.[25]

The Hottentots are generally thought to be closely related to the Bushmen on certain morphological and linguistic grounds, but substantial blood-group differences between them are known,[26,27] and it seems that the haptoglobins may be another point of distinction. It is doubtful whether any sizeable Nama-speaking Hottentot groups survive who are free from European ancestry (my estimate is about 2,000 individuals in the Richtersveld and adjacent regions). Intermixing with Dutch, German, and English has led to the formation of various 'hybrid' Coloured communities. From what is known of European haptoglobin frequencies, however, this would have made them more like the Bushmen in this respect, so that the earlier Hottentots may have been more distinct. The effect of intermixture with the Malays, brought to the Cape of Good Hope as skilled artisans in the 18th century, cannot at present be assessed. There is probably extremely little Malay admixture in those Cape Coloureds tested. It is interesting to note that in Malaya the Malays exhibit a gene frequency of 0·24 for Hp^1, which is slightly lower than that (0·28) of the Malayan Chinese, but significantly higher than the 0·09 of the Malayan Indians.[17] Asiatic Indian students studying in the USA have a frequency of 0·18.[14]

A slow transferrin variant was found to be quite common in the Bushmen, and one serum which showed this variant alone was identified (by Dr. O. Smithies) as βD_1 which was first described by Smithies[28] in Australian populations and American Negro sera, and is fairly common in some West African populations.[29] The variant is evidently less frequent in the Zulus and Cape Coloureds, and perhaps in the Hottentots, than in the Bushmen.

The distribution of the haptoglobins in a small region of West Africa shows a marked discrepancy for the frequency of haptoglobin deficiency or O-O, as indicated in the results of Allison *et al.*,[13] on the one hand, and Sutton *et al.*[21] on the other. In one-third of 99 Yoruba studied by Allison *et al.*,[13] haptoglobin was deficient. Most of the remainder were type 1. Sutton *et al.*[21] did not report any ahaptoglobinaemia in their series from Liberia and the Ivory Coast. In a subsequent study, Sutton *et al.*[14] found that the presence of considerable haemolysis interfered with the detection of 1 - 1, so that their diagnosis of 1 - 1 was usually based on a negative criterion, namely, the failure to demonstrate the type 2 - 1 or 2 - 2 pattern with benzidine staining. They state that probably some of these 1 - 1 individuals actually do not have detectable amounts of haptoglobin. For this reason their frequencies of Hp^1 (in their survey) represent maximum values. Allison *et al.*,[13] in comparing their data with the absence of ahaptoglobinaemia in the 1956 study[21] of Sutton *et al.*, attributed it to inclusion of group O-O in the 1 - 1 group, because the distinction between these two groups is difficult without specific staining of haemoglobin. However, if one takes Sutton *et al.*'s results[21] and assumes an approximate one-third of ahaptoglobinaemics, the frequency for Hp^1 would be reduced from 0·70 and 0·72 to 0·54 and 0·52, which brings the Ivory Coast and Liberia Negroes into line with Zulus and Venezuelans, and would bring their frequency lower than that of US Negroes, Apache Indians, and Micronesians. It would appear that the Hp^1 frequency, as seen in the Yoruba, has not been affected by the Caucasoid dilution in the US Negroes. The highest incidence of haptoglobin deficiency, other than in the Yoruba and Sutton's third series of Liberians, is only 4·2% in US Negroes, out of more than 33 world-wide surveys reported, in which 13 out of 33 displayed no ahaptoglobinaemia, and in the remainder the mean is less than 2% per population group.

It must be interesting to break down all the West African data to tribal distribution, and study their inter-relationships afresh. Certainly the significance of a 32% haptoglobin deficiency in the Yoruba is not at all obvious. Allison *et al.*,[13] in noting the difference between this figure and Sutton *et al.*'s data[14] for Liberia and Ivory Coast, stated that the discrepancy may represent a difference between Africans from these different regions, but they excluded this in preference to an explanation of difference in laboratory techniques. However, it should be noted that the haemoglobin *S* frequency in Liberians is relatively low compared with that in Nigeria, and that the haemoglobin *C* frequency is relatively low in Nigeria and in Liberia, and about the same cline. Whatever produces the discrepancy in O-O, it is not a constant in relation to abnormal haemoglobins. Haptoglobins are not present in most newborns,[9] but absence is unusual after the age of 4 months. Deficiency in the adult represents either a persistence of what could be called the foetal state, or a secondary loss from various causes. If haptoglobin deficiency is related to malaria somehow, one would expect a close correlation with high frequencies of sickle-cell homozygotes on the basis of the theory that the heterozygotes constitute an advantageous selection.

Ahaptoglobinaemia may occur for several reasons. Laurell and Nyman[30] have shown that, if the circulation is saturated with haemoglobin, as in acute haemolytic conditions, the plasma can be completely depleted of haptoglobins in 24 hours. It is possible that chronic haemolytic conditions, so common in Africa, may produce a similar picture. In the Japanese population of Sapporo, studied by Matsunaga and Murai,[16] 4 of the 5 haptoglobin-deficient cases had no history of anaemia or liver disease. The 5th had a history of hepatitis. They also found haptoglobin deficiency in 4 patients with liver cirrhosis, one case having liver metastases. Haptoglobins were also scarcely recognizable in 1 of 3 patients with hepatoma. Haptoglobin deficiency has also been observed in European and American Negro groups in whom there was neither evidence of a haemolytic condition nor a history of malaria. With respect to haptoglobin synthesis and the gene or genes controlling this, it is worthy of note that the liver in the African Negro has a rather unique metabolism (whether inherent or acquired, or both, is not certain), but African Negroes very commonly suffer liver cirrhosis and for some reason, probably related, have a high incidence of primary carcinoma of the liver, which is rare in other racial and geographic groups.

Giblett[31] pointed out that inheritance studies of ahaptoglobinaemia have so far been inconclusive. However, in some families, both parents of a subject with haptoglobin deficiency appear to carry a pair of normal haptoglobin genes. Thus it is clear that if a mechanism is involved, the responsible gene cannot be an allele at the Hp locus, nor can it be a Mendelian dominant. It has been suggested that the Hp O-O phenotype may be due to a modifying gene at a separate locus, or alternatively represents the homozygous condition for the Hp^{2M}. Thus it seems that in haptoglobin

deficiency one may encounter a primary, genetically-determined ahaptoglobinaemia and/or a secondary, acquired, disease-produced and temporary ahaptoglobinaemia. A solution to the significance of this phenomenon will only become possible when a laboratory-tested distinction between the two forms may be elicited. The inherited form does not appear to be controlled by alleles at the usual haptoglobin locus.[12,14] Some ahaptoglobinaemics possess small amounts of normal haptoglobin.[14]

It has been suggested[13] that the true polymorphism exhibited by the haptoglobins may have been maintained, like those of the blood groups and abnormal haemoglobins, by a balance of selective forces related to susceptibility to disease. They state that the absence of haptoglobins may be disadvantageous, particularly in regions where there is a deficiency in the diet. That so many Yoruba lack haptoglobins is presumably due to a compensatory advantage in persons heterozygous for the factor concerned. The low frequency of type 2 - 2 in the tropics was felt to confer protection against one or more conditions common in temperate, but not in tropical climates. However, the evidence available today does not obviously confirm these hypotheses, which are based on insignificant sampling of tropical areas, because other tropical areas also give high frequencies of type 2 - 2. It is clear that correlative studies with distributional incidence of schistosomiasis, yaws, onyalai, malaria, kalar-azar, helminthiases, etc. may provide some clue to selective advantage, but on the present evidence this is quite imperceptible.

Comparative anatomical studies have not yet helped in determining a positive direction towards the nature of the selective advantage of one or other form of haptoglobin in maintaining the balanced polymorphism. A number of investigations on non-human primates[33,34] indicate that all the animals studied had an electrophoretic band in the approximate position of haptoglobin 1 - 1. None had anything resembling the human 2 - 1 or 2 - 2. It would appear that type Hp1 - 1 is the primary or primitive type from which present types have mutated, and that a mutation occurred after *Homo* or his immediate ancestors had moved away from the Pongid line. The gene's favourable spread into various populations was limited by various factors, as yet unknown. It is at present not possible to assess whether such a mutation actually promoted hominid evolution in one or other direction. It appears reasonable to assume that the possession of the Hp^1 gene is an advantage among hominoids, at least.

TABLE III. RACIAL (ETHNIC) GROUPING ACCORDING TO FREQUENCY OF ALLELE Hp^1

Hp^1 frequency	
<0·25	Malays, Asiatic Indians, Japanese
0·26 – 0·40	Swedish Lapps, Finns, Italians, Bushmen, Chinese, Alaskan Eskimoes, Danes, French, Swedes, US Caucasoids, Spanish Basques, British (Oxford), Norwegians, Pygmies (Congo)
0·41 – 0·55	Anaktuviak Eskimoes, Athabascan Indians, Canadian Caucasoids, Venezuelans (Caracas), British, North Italians, Cape Coloured, Zulus, Xhosa and Msutu
0·56 – 0·70	Apache Indians, U.S. Negroes, US Caucasoids, Congolese, Hottentots, Micronesians (Marshall Islands)
>0·70	Liberia and Ivory Coast Negroes (mean), Yoruba (Nigeria)

If one takes the range of variation of Hp^1 within the Liberian and Ivory Coast tribes,[14] and uses this range arbitrarily for dividing up the racial and ethnic populations studied in respect of Hp^1, one finds a rather curious grouping, the significance of which is not apparent. But it should make us wary of the pitfalls inherent in over-hypothesizing and over-emphasizing inadequate data.

In conclusion, it seems that in the study of haemoglobin-binding proteins (haptoglobins), as well as the iron-binding serum proteins (transferrins), we have a rather useful genetic mechanism for marking out racial and ecologically-induced differences, and for indicating possible selective adaptations in man. The usefulness of such studies cannot yet be assessed in Africa because of the inadequacy of the available, limited data.

Grants from the Wenner-Gren Foundation for Anthropological Research, Inc., New York and the Dr. C. Herman Research Fund, University of Cape Town, made it possible for the field work to be carried out.

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SIMULTANEOUS BILATERAL PULMONARY RESECTION

R. P. HEWITSON, F.R.C.S. (ENG. and EDIN.)
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In the surgical treatment of any condition it is more satisfactory to achieve the desired result in one procedure rather than two, provided that the operative risk, both in mortality and morbidity, makes this justifiable. With developments in medical practice, various operations, which formerly were performed in two stages, are now completed in one. For the patient this is preferable, both psychologically and economically. It saves theatre time and other hospital time. Thus, the possibility of operating simultaneously on both lungs has been raised when the disease process demands bilateral surgical action.

The practicability of an operation of this nature was demonstrated in 1952 by Overholt,[1] and by 1956 he was able to report on 10 cases.[2] Lewis *et al.*[3] mention that in 1882 Block attempted a bilateral lung resection, but it was not successful. In this report they cite 16 cases of their own, and a year later Björk[4] reported on 4 patients on whom he had combined bilateral resection with a unilateral osteoplastic thoracoplasty at one sitting. The only other report noted of simultaneous bilateral resection was published by Baronofsky *et al.*[5] in 1957; the resection was carried out in dealing with spontaneous pneumothoraces. Since 1956 isolated reports have appeared from other countries in languages other than English, e.g. from Holland,[7] France,[8] Brazil,[9] and Russia.[10]

A comparable procedure is a resection of part of the remaining lung after total pneumonectomy, which has been shown to be a reasonable operation in certain circumstances.[6]

In the reported cases there have been 3 indications for surgery, i.e. bronchiectasis, tuberculosis, and spontaneous pneumothorax. The fact that Overholt[2] could only report on 10 cases after 4 years indicates how uncommonly the opportunity arises. Most of his cases were treated for bronchiectasis.

Lewis *et al.*[3] were stimulated to attempt simultaneous surgery by the number of bilateral cases of pulmonary tuberculosis presenting from a large mental institution. After unilateral thoracotomy and resection it takes some 4 - 8 weeks before lung function begins to approximate to its final figures, and maximal improvement needs 4 - 6 months according to Pecora.[13] Consequently, it has been customary to wait a few months before the second operation is performed in a staged bilateral resection. This staging presents considerably increased difficulties in insane patients, so that Lewis *et al.*[3] tried operating on the two sides simultaneously and found that it was tolerated very well.

Shumway *et al.*[11] have suggested that simultaneous bilateral resection actually results in better function of the remaining lung tissue than if the operations are staged, because of greater stimulation to use the lungs. This seems doubtful even in theory, since there is a considerable temporary load placed on the non-operated lung during unilateral surgery. Pecora[12] disputes this suggestion and has tried to produce some factual evidence. However, his figures include only one case of simultaneous resection, and the vital capacity was the only function test used for comparison. He maintains that there is no functional benefit from the simultaneous procedure and that the final ventilatory deficit appears to be the sum of each resection, whether simultaneous or staged.

An argument can be made for staging the resections in patients with poor pulmonary function, so that the first side can recover as much as possible before the function of the opposite side is temporarily reduced in a similar way. Against this must be weighed the advantage of removing all the disease at one sitting, so that the risks of postoperative bronchial spread of infection or a tuberculous flare-up are reduced to a minimum.

Usually cases have been accepted for simultaneous surgery only when small amounts of lung tissue need removal, but Björk,[4] for instance, has shown that much more can be removed at one sitting than would previously have been thought possible. A great deal depends on the methods and facilities available for postoperative respiratory assistance. Thus Björk[4] has removed in one patient the right upper lobe, the whole left upper lobe, and the apical segment of the left lower lobe; combining this with an osteoplastic thoracoplasty on the left. The degree of destruction of the removed lung tissue and the function of the remaining part are important factors.

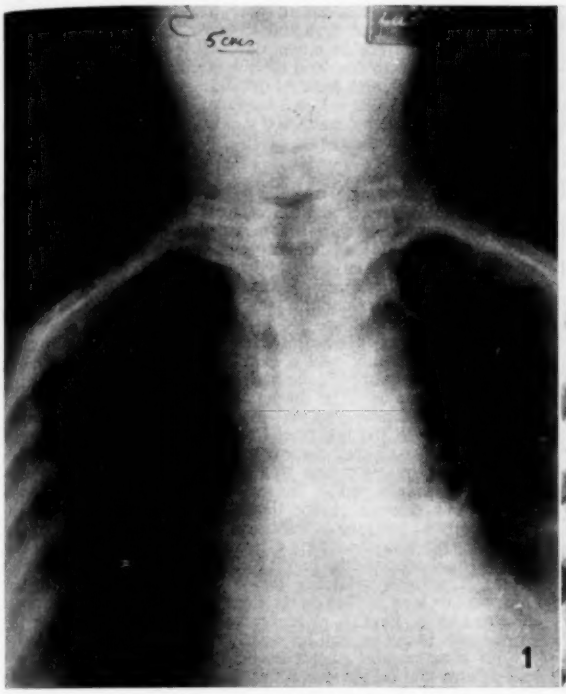
Even in tuberculosis the indications for bilateral resection are not common. Bilateral disease so often means widespread involvement with correspondingly reduced pulmonary function. The largest reported series is probably that of Curtis *et al.*,[14] who had 87 patients for bilateral surgery out of a total of 450 patients for resection. The majority were segmental resections with an average loss of function after the two operations of about 14% of the pre-operative figure. Where lobectomy was the procedure on both sides (7 cases), the loss was about 27% of the pre-operative figure. (Tests for vital capacity and maximum breathing capacity were used.) Cooley *et al.*[15] had 12 out of 189 patients for resection, and they stress that assessment must be careful since 3 of their 12 patients fell into the 'failure' category. These were all staged resections.

Of the cases of simultaneous resection reported in the English literature, there has been no mortality.

Selection of Cases

The less lung tissue needing resection and the better the function of the remaining lung tissue, the better is the case suited to simultaneous bilateral surgery. The indication for removing a particular portion of lung should be the same as when contemplating unilateral surgery or staged bilateral surgery. However, smaller lesions are, generally speaking, more suited to a simultaneous approach than larger ones. Extensive fibrosis or emphysema are factors against bilateral surgery, though, as mentioned above, Björk[4] has had success with the removal of up to 9 pulmonary segments. The removal of whole lobes rather than segmental resection has the advantage that the postoperative air leak may be minimal, but, with adequate care this factor can be overcome and should not influence the decision.

The patient's general condition should be as good as possible. This may entail much pre-operative care, with antibiotics, diet, and physiotherapy. Tuberculous patients should preferably be ambulant.



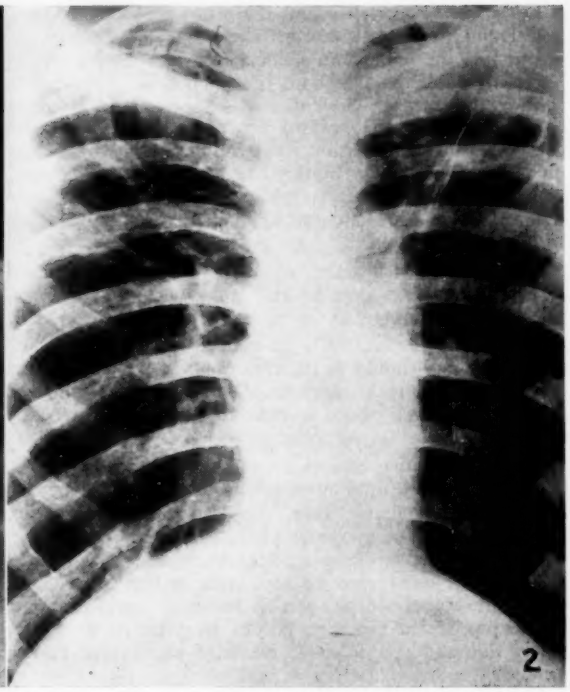


Fig. 1. Tomogram at 5 cm. showing bilateral apical cystic cavitation. *Fig. 2.* Postero-anterior chest X-ray showing tuberculous cavitation in both upper lobes and emphysema of the left lower lobe.

The patient should be young, if possible. However, Lewis *et al.*,[3] who make this point, nevertheless found that the average age of their 16 patients was 44 years, with a range of 25 - 56 years. Thus age alone is no absolute criterion, but is a partial guide to the state of the rest of the cardio-pulmonary system.

Mental patients apparently afford a special indication for simultaneous resection in tuberculotics, since nursing is easier and it is better in a mental institution if all the disease can be eradicated at an early date to prevent spread of infection. Mentally disordered patients often cannot be trusted in dealing with sputum or in other normal precautions.

Pulmonary-function tests play some part in assessment as in unilateral cases, but they have their limitations. Low figures do not necessarily exclude surgery.

In bronchiectasis the decision is often very difficult, since it is precisely in the bilateral case where there may be difficulty in being completely sure of the limits of the disease and where one is hesitant to recommend even staged resection. Bilateral bronchiectasis is so commonly a disease which starts insidiously in youth without much associated atelectasis, that one is reluctant to remove large areas of functioning lung tissue. Again, sometimes there is sufficient improvement after unilateral resection in these patients to decide against operating on the second side. These various factors make one particularly cautious in cases of bronchiectasis. One other point which may influence the decision slightly is that there is usually some bronchitis in the non-dilated bronchi of these patients, and this can make the postoperative course more difficult than, for example, in well-drained upper-lobe tuberculosis where there may be little sputum.

Operative Considerations

The pre-operative care is the same as for unilateral surgery, but must be attended to meticulously. Thus, the amount of sputum must be at a minimum, tuberculosis control by chemotherapy must be at its maximum, and the general health should be as good as possible.

There are 2 possible operative approaches: from the front or from the back. Overholt[1] has pioneered the prone position for pulmonary surgery and he advocated this for simultaneous bilateral resections also. Others, such as Björk[4] follow this method and he makes 2 points about it: the surgeon can be content with dealing with only one side if factors arise to dictate this, without opening the opposite side of the chest at all; and it is by far the easier position if a thoracoplasty is needed at the same time.

The supine position is used by Lewis *et al.*[3]; they split the sternum, but this is not always necessary. The 2 sides may be approached from the front through separate incisions. The table may be tilted from side to side to assist, if necessary. If the sternum is split, there is the danger of exposing the mediastinum to the infection, either tuberculous or pyogenic, for which the operation is being done. It is noteworthy that in their 16 reported cases, there were 3 with sternal separation as a major complication. This occurred in mental patients. Lewis and his co-workers[3] suggest that mentally disordered patients with reduced regard for pain, may well throw an additional strain on the sternal wound during convalescence.

In the cases reported below, the prone position was used and found to be satisfactory. There was no difficulty in positioning the patient on sandbags in the absence of an 'Overholt' table, so that both upper arms were dependent to draw the scapulae forward. It was not necessary to move the patient between the two operative sides. The usual curved periscapular incision was used, and the two incisions did not meet medially. Björk[4] used an inverted Y incision, and some sloughing occurred in one case where the angle was too acute. An intercostal approach is usually adequate without rib resection.

A double-lumen intratracheal tube for separate inflation of each lung, such as a Carlen's tube, is useful, but not essential. It was only used in one of the cases reported here.

The method of resection is the same as is usually followed, but special care should be taken to reduce the air leak from raw lung surface to a minimum. At the close, the drainage tubes must be well placed to deal with both apical air and basal drainage; at least 2 tubes on each side are essential.

The postoperative care is a vital part of the treatment; it consists mainly of a meticulous adoption of the usual methods for a unilateral case. The need for sedation is no more than usual and must allow full cooperation in coughing and physiotherapy. Blood replacement must be adequate. The drainage tubes must be kept open so that the pleural spaces are obliterated as soon as possible. Suction on the underwater sealed drainage bottles is often necessary to ensure this and also to avoid excessive paradoxical movement.

Any indication of respiratory inadequacy must be dealt with promptly. Poor coughing out of secretions, poor respiratory movements, and dyspnoea caused by lack of lung tissue, point towards early tracheotomy, which in some cases may be advisable on the operating table. Tracheotomy reduces the respiratory dead space, thus reducing the amount of movement needed, and it allows free suction of secretions at all times. If this alone is insufficient, positive-pressure assistance may be needed. Nevertheless, such patients are not necessarily respiratory cripples later, once the remaining lung has adjusted itself

CASE REPORTS

Case 1

F.E., a Coloured female aged 19 years, had been treated for some 9 months for bilateral upper-zone tuberculosis. She was left with 'cystic' cavitation at both apices (Fig. 1), and bronchography showed that the bronchial damage involved the whole right upper lobe and the apico-posterior segment of the left upper lobe.

At operation, the right side was resected first. The lobe was firmly adherent and there was virtually no lesser fissure, so that a segmental type of strip was needed. The raw surface was not oversewn and there was little air leak. On the left side the segment stripped off readily, but the raw surface was oversewn—the edges fell together readily.

Two tubes were left in each pleural space, and it was possible to remove them all by the 4th day, after which the patient was ambulant. No particular postoperative treatment was needed in this case.

The pre-operative function tests for this patient showed a forced expiratory volume of 1,500 c.c. in 1 second and 2,000 c.c. in 3 seconds.

Case 2

M.L., a Coloured female aged 16 years, was similar in many ways to the previous patient. The same type of pathology was present, but was confined on the right to the posterior and apical segments, and on the left to the apico-posterior segment. The left side was resected first and it was not necessary to oversew the raw lung surface on either side. All four drainage tubes were removed by the 4th day, and there was no postoperative difficulty.

The function figures were as follows: pre-operatively, 1,400 c.c. in 1 second and 1,800 c.c. in 3 seconds; 6 months postoperatively, 1,300 c.c. in 1 second and 1,600 c.c. in 3 seconds. Thus the functional loss was in the region of 10%.

Case 3

S.S., a Coloured male aged 25 years, had been under treatment for 2 years for pulmonary tuberculosis, but his sputum was still persistently positive with organisms that were resistant to the ordinary drugs. He had not responded to cycloserine. The X-ray film showed cavitation in both upper lobes with much emphysema at the left base (Fig. 2). His forced expiratory volume for 1 second was 1,000 c.c. Pyrazinamide was given for 3 weeks before and 4 weeks after operation.

The right upper lobe was removed with a difficult strip along a fused lesser fissure and a partly extrapleural dissection. On the left the whole upper lobe had to be resected since the lingula was diseased. In view of the emphysema of the lower lobe, an osteoplastic thoracoplasty of the Björk type was performed, from the 2nd to the 6th ribs. Finally, because of the poor pulmonary function, a tracheotomy was done on the table.

The postoperative course was fairly smooth and the tracheotomy tube was removed on the 3rd day. However, on the following day there was some gastric distension and the patient became very dyspnoeic; vigorous treatment was needed to relieve this. Sedation and bronchodilator drugs were required for some time after this episode, and he had 2 further minor attacks of a similar nature. The other retarding factor was a persistent small leak from the raw lung surface on the right for which the apical drainage tube had to be kept in place for some 3 weeks. Soon after this he was able to get up and he rapidly recovered his mobility.

Four months after operation he could produce 700 c.c. in 1 second and 900 c.c. in 4 seconds. In spite of these poor figures he was ambulant, with a negative sputum.

Case 4

M.S., a Coloured female aged 15 years, had been treated for some 18 months for tuberculosis and was left with bilaterally destroyed upper lobes, the left having a small cavity. The sputum was negative at this stage. Both upper lobes were well shrunken so that, although the forced expiratory volume for 1 second was only 1,200 c.c., it was decided to remove both upper lobes at the same operation.

No space-reducing procedure was needed and tracheotomy was not done. Recovery was entirely uneventful.

Case 5

E.L., a Coloured male aged 24 years, was assessed in 1958 for a chronic productive cough and the bronchogram revealed bronchiectasis of both bases, concentrated mainly in the right middle lobe, the left lower lobe, and the lingular segment of the left upper lobe. Physiotherapy was advised and he was referred to the ENT department where radical maxillary antrostomies were performed. He was followed-up as an outpatient, but the constant purulent sputum was a real trial to him. Check bronchography, 2 years after the first, showed much more atelectasis of the left lower lobe, and although the basal bronchi on the right were not perfectly normal, it was decided to resect the right middle lobe and the lingular segment with the left lower lobe.

At operation no clear lesser fissure was found on the right and there was a fair amount of postoperative air leak from this strip. On the left the segmental strip was reasonably easy and the raw surface was not oversewn.

On the day after operation there was a fair amount of secretion which he could not cough out, and tracheotomy was performed. The tube had to be kept in until the 10th day because of his inability to raise the mucoid sputum even though it was thin. The air leaks continued for longer than one would normally expect in a unilateral case. On the right the tubes were removed by the 8th day, and on the left a basal tube was kept in for 6 weeks.

This was not an ideal case since the operation has unfortunately not cleared all his bronchiectasis. A great deal of the difficulty was probably due to the associated bronchitis with its secretions, in contrast with the tuberculous cases which were mainly dry.

SUMMARY

The possibility of performing resection on both lungs at the same operation is discussed, and the need for and advisability of a procedure of this nature are mentioned, with a brief review of the literature.

It is pointed out that there are few indications for performing simultaneous bilateral resection, the main two being tuberculosis and bronchiectasis.

The operative approach and the particular postoperative needs are discussed.

Finally, 5 cases are reported (without mortality) to illustrate the method. Four of these patients suffered from tuberculosis and one from bronchiectasis.

Thanks are due to the nursing and medical staff who have been essential to the teamwork which is necessary for such operations; also to the Superintendents of the Brooklyn Chest and Groote Schuur Hospitals for permission to publish; and to Mr. B. Todt for the radiological reproductions.

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From the Secretary's Desk

Medical Protection

In all forms of insurance it can be a great shock to find that one's policy has lapsed at a time when a claim is made. This happened recently to a member when a patient lodged a claim against him for damages, and it was pointed out to him that he had neglected to pay his dues some nine months previously. Fortunately the outcome for our member is satisfactory, but it could so easily have been otherwise. The onus is on the insured person to see that his premium or subscription is paid, and it would be as well for every member to check his own payments to see not only that he is covered, but that he is adequately covered.

Recently we had reason to send additional reminders to a number of members of the Medical Protection Society in the form of registered letters, pointing out that they had ceased to be covered for some time since their subscriptions had remained unpaid in spite of reminders. It is extraordinary how many have not yet replied and remain unprotected. Unfortunately one never knows when trouble may arise.

43rd South African Medical Congress

Preparations for the Congress in Cape Town in September are going ahead and it would seem that, from all angles, the week will be both profitable and a pleasure for all who are able to be present. Those who have not yet sent in their intention cards are urged to do so as soon as possible.

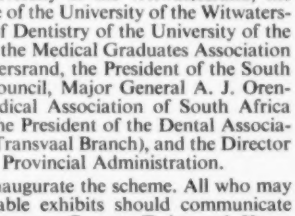
Benevolent Fund

On 10 May, I was privileged to attend a dinner-dance in Port Elizabeth organized by the Medical Wives' Association of the Cape Midland Branch. This successful function was held in the Lotus Garden Restaurant and Chinese dishes were served. It was well attended and proved most enjoyable.

The medical wives of the Natal Coastal Branch are at present organizing, through Messrs. Lindsay Smithers (Pty.) Ltd., a competition for a Triumph Herald motor car in aid of the Benevolent Fund. It is hoped that tickets for the competition will be bought by members throughout the country. Many of the Branches have already indicated their willingness to help by the sale of books of tickets, and the Management Committee of the Benevolent Fund is anxious that this effort should meet with the success it deserves. At present the Fund is helping to support some 36 widows and children, and additional calls are continually being made on the Fund.

Unethical Behaviour

At the recent meeting of the Executive Committee of Federal Council certain examples of unethical behaviour were mentioned which had recently come to the notice of members. Some of these were worse than unethical, being frankly dishonest. It was realized that it is difficult for young and inexperienced practitioners to resist the pleas of their patients under the threat of losing them; but it was decided that it should be pointed out to those who might be so tempted that serious trouble could result for them if certificates were supplied for medical aid, medical insurance, or for any other purpose, which contained incorrect dates and wrong statements. Older practitioners are asked to keep a watch on practices of this nature and to help their younger colleagues.



Medical House
Cape Town
9 June 1961

MUSEUM OF THE HISTORY OF MEDICINE

MEDICAL GRADUATES ASSOCIATION, UNIVERSITY OF THE WITWATERSRAND, JOHANNESBURG

The Medical Graduates Association has agreed to sponsor a scheme for the establishment of a Museum of the History of Medicine in the Medical School of the University of the Witwatersrand. It is proposed to collect and preserve for permanent record all material which illustrates the history of medicine in general, and its development in South Africa in particular.

These exhibits will include instruments, apparatus, books, copies of original papers (if possible with the signature of the author), photographs, etc.

It is hoped to house this museum in the New Medical School Library.

The following have graciously accepted patronage of this venture:

His Worship the Mayor of Johannesburg, the Vice-Chancellor and Principal of the University of the Witwatersrand, the Chairman of the Council of the University of the Witwatersrand, the President of the Convocation of the University of the Witwatersrand, the Dean of the Faculty of Medicine of the University of the Witwatersrand, the Dean of the Faculty of Dentistry of the University of the Witwatersrand, the President of the Medical Graduates Association of the University of the Witwatersrand, the President of the South African Medical and Dental Council, Major General A. J. Orenstein, the President of the Medical Association of South Africa (Southern Transvaal Branch), the President of the Dental Association of South Africa (Southern Transvaal Branch), and the Director of Hospital Services, Transvaal Provincial Administration.

It has now been decided to inaugurate the scheme. All who may be interested in donating suitable exhibits should communicate with: Dr. Cyril Adler, 701 Ingrams Corner, Twist and Kotze Streets, Hospital Hill, Johannesburg (telephone 44-1938).

Where possible the following should be furnished with each exhibit: (1) Name of donor, (2) description of exhibit, and (3) brief description of exhibit.

IN DIE VERBYGAAN : PASSING EVENTS

Dr. C. L. Botha, radioterapeut, van Pretoria het sy adres verander na Robert Kock Mediese-gebou 136, Pretoriusstraat, Pretoria. Die telefoonnommer bly onveranderd.

Dr. C. L. Botha, radiotherapist, of Pretoria has changed his address to 136 Robert Kock Medical Building, Pretorius Street, Pretoria. The telephone number remains unchanged.

* * *

Dr. Wynne Rigal, M.B., Ch.B., who graduated from the University of Cape Town in 1955 and proceeded to Oxford in 1957 as a Rhodes Scholar, has been awarded the degree of D.Phil. (Oxon.) for his thesis on 'A study of bone development using tissue culture as the main technique'; and has passed the final examinations of the Royal College of Surgeons of Edinburgh. Mr. Rigal is now the Orthopaedic Registrar at the Royal Infirmary and Margaret Rose Orthopaedic Hospital, in Edinburgh.

South African Institute for Medical Research, Johannesburg, Staff Scientific Meeting. The next meeting will be held on Monday 3 July at 5.10 p.m. in the Institute Lecture Theatre. Dr. L. Schrire and Mr. D. H. S. Davis will speak on 'An outbreak of plague in South West Africa'.

* * *

Dr. Joseph B. Herman of Cape Town, has been invited to present a paper at the International Diabetes Congress in Geneva. Dr. Herman leaves for Geneva on 7 July 1961 and will also attend the Medical Congress in Israel.

* * *

Dr. F. Benjamin, of Port Elizabeth, was admitted to the Fellowship of the Royal College of Obstetricians and Gynaecologists at the meeting of the Council held in London on 27 May 1961.

FARMASEUTIESE NUUS : PHARMACEUTICAL NEWS

IN MEMORIAM

MR. GEORGE PRESTON WALLACE

We regret to announce that Mr. George Preston Wallace, at the age of 51, passed away at his home in Rondebosch, Cape, on 31 May 1961, on the eve of a projected overseas visit to Europe, Britain and America.

He will be remembered by the medical profession for his untiring efforts to further the advancement of new therapies and exact knowledge of these under the auspices of J. R. Geigy (S.A.), of Switzerland, in South Africa.

Mr. Wallace came to South Africa in 1938 as sales representative for Glaxo Limited of England. In 1942 he undertook the management of Petersen Limited in Cape Town as Managing Director and was on the Johannesburg Board of Directors of the same company. In 1947 he was responsible for the formation of McDonald Wallace & Co. (Pty.) Limited, and in 1949 of Pharmakers (Pty.) Ltd., with head offices in Cape Town, for the distribution to the medical and pharmaceutical professions and industry of high class pharmaceuticals and chemicals. In addition to these activities he was, three years ago, appointed Chairman of Geigy South Africa (Pty.) Limited. This firm is concerned with the distribution of the parent company's dyestuffs, chemicals and insecticides in Southern Africa.

Mr. Wallace will be missed by all who came in contact with him as a good friend, and in business a man of high moral standards and ethics, with an understanding of human nature and great tolerance.

BOEKBESPREKINGS : BOOK REVIEWS

REFLEXES

The Discovery of Reflexes. By E. G. T. Liddell, D.M., F.R.S. Pp. 174. 23 illustrations. English price: R3·00. Oxford: Clarendon Press. 1960. South African agents: Oxford University Press, P.O. Box 1141, Cape Town.

This book is, in effect, a tribute to the life and work of Charles Sherrington, a task for which the author is particularly fitted by reason of his own distinction as a neurophysiologist and as one of Sherrington's distinguished pupils.

It is a happy fact that many of those who worked with Sherrington are able to write extremely well, almost as though they had acquired in their contacts with him some of his own great talent in this sphere. Professor Liddell tells a fascinating story of the history of the knowledge of the nerve cell from the time in 1665 when Robert Hooke gave the earliest description. He follows over the years, with the improvement in the microscope and in the techniques of preparing specimens, the development of more modern concepts with the last word yet far from being said. Similarly, with nerve conduction and the ideas of the electrical phenomena of nerves—the early theories and experiments, and so to 1879, the year when Sherrington, a young student of 21, entered Cambridge and began to work on reflex action of the nervous system culminating in the classic 'Integrative action of the nervous system' published in 1906.

Let me hasten to reassure those who may think this a dull historical work; the author is a brilliant teller of a fascinating story, and once started it is a book that everyone will read avidly to the last page. To all interested in neurophysiology this is a classic to buy and cherish; for the everyday doctor, and especially for those who ever venture into print, it is an example of how to write well about science. S.B.

MEDICAL RESEARCH

Methods in Medical Research. Vol. 8. Edited by H. D. Bruner. Pp. xiv+368. 39 figures. 89.75. Chicago: Year Book Publishers, Inc. 1960.

This volume maintains the same high standard of its predecessors and is of particular interest to research workers.

The present number is concerned with 3 main subjects: (1) The life history of the erythrocyte, (2) the measurement of responses of involuntary muscle, and (3) peripheral-blood-flow measurement.

The first section commences with a discussion of the examination of the bone marrow followed by an account of bone-marrow culture and the difficulties of interpretation. The reticulocyte count has the merit of simplicity, but observations show it may be unreliable, since increases in reticulocytes need not necessarily be accompanied by increases in peripheral red-cell counts nor in the number of nucleated red cells in the marrow. Radio-iron methods are fully described and the underlying assumptions in using Fe^{59} for determining iron turnover have been analysed. Another method of determining bound iron and unsaturated iron-binding capacity of plasma is reported by Schade. Methods of measuring red-cell volume (P^{32}, Cr^{51}, Fe^{59}) are considered and the difference between red-cell volume determined by isotope-tagging methods as opposed to carbon monoxide, is suggested by Root and Allen. Of the methods employed for studying the life span of the red cell, the best probably embodies the use of N^{15}- or C^{14}-labelled glycine but other methods are considered. The metabolism and methods of estimation of urobilinogen are fully described, and although urobilinogen excretion is an imperfect index of red-cell destruction, it still plays a useful rôle in clinical medicine.

The second section on measurement of responses of involuntary

muscle contains a useful account of the investigation of gastro-intestinal motility.

The third section has a very complete discussion of peripheral-blood-flow measurement including measurement of the hepatic, coronary, and renal blood flow (blood-tissue exchange methods). Electro-mechanical methods including venous occlusion plethysmography, calorimetry, the use of Bristle and the Square-wave electro-magnetic flowmeters, are considered in detail.

For workers in these fields, this book may be strongly recommended. L.E.

YEAR BOOK OF OPHTHALMOLOGY

The Year Book of Ophthalmology, 1959 - 1960. Edited by William F. Hughes, M.D. Pp. 406. 95 figures. $8.00. Chicago: Year Book Publishers, Inc. 1960.

After ten years' editorship of the Ophthalmology Year Book series, Derrick Vail has been succeeded by William F. Hughes. The present volume maintains the high standard set by its predecessors and is assured of many more years of support.

The introductory special article on clinical electroretinography is of particular significance in the light of the recent visit of Prof. Jules Francois to this country. There follows, under appropriate chapters, a selection of the best articles appearing in world literature over the previous twelve months. These articles are adequately summarized, are of fair length and accompanied, as before, by critical editorial comment, which is particularly required when contrasting views are presented, such as the mechanical and neurovascular theories of the aetiology of glaucoma.

This book continues to be the best summary of the year's literature. Previous readers of the Year Book will undoubtedly add this volume to their libraries, and new readers can only derive benefit and instruction from introduction to it. L.S.

CANCER

A Clinical Prospect of the Cancer Problem. Introductory Volume. Edited by D. W. Smithers, M.D., F.R.C.P., F.F.R. Pp. xv+232. Illustrated. R3.75+21c postage abroad. Edinburgh: E. & S. Livingstone Ltd.

This introductory survey to a series of monographs on 'Neoplastic disease at various sites', written by the general editor, is produced to form a philosophic background for the succeeding volumes.

While intentionally speculative and provocative, it is refreshingly stimulating. The reader is led away from old familiar paths of conventional approach, from the masses and the 'cancer cells' that tend to constitute our more habitual thought-forms, to alterations in pattern, disorganized behaviour and to the concept that 'cancer is a word for a selection of extreme behaviour patterns within the class of tissue malformations being normally contained within the subdivision tumours'.

The discussions on the natural history of neoplasia, the theories of causation, and the influences of hormones and other relevant topics add very considerably to the general value of this production.

This outstanding contribution is worthy of serious study by all. It augurs well for the whole series. J.M.G.

GYNAECOLOGY

Text-book of Gynaecology. 5th edition. By J. H. Peel, M.A., B.M., B.Ch. (Oxon.), F.R.C.S., F.R.C.O.G. Pp. 491+xv. 209 figures. R3.00 net. London: William Heinemann Medical Books Ltd. 1960.

The fifth edition of this well-known textbook is fully up to the standard of its predecessors. John Peel's textbook is widely used by undergraduate students both in this country and overseas, and admirably suits the purpose. The recent developments in gynaecology are included in this new edition; as reflected by general revision throughout and the incorporation of a new chapter on intersex.

While the standard throughout is high, particularly good chapters are those dealing with amenorrhoea, menorrhagia, gynaecological endocrinology, and general treatment in gynaecology.

Although the author wrote the book primarily for undergraduate students, the basic facts and recent advances are so well handled that it can confidently be recommended to general practitioners. H.M.

NEOPLASTIC DISEASE OF BONE

Neoplasms of Bone and Related Conditions. 2nd edition. By Bradly L. Coley, M.D. Pp. xv+863. 649 illustrations and 31 tables. $30.00. New York: Paul B. Hoeber, Inc. 1960.

This is a beautifully produced, but rather heavy and expensive book of reference on tumours of bone. It is very clearly set out and easy to read. The bibliography is rather incomplete for such a specialized work.

The wisdom of including sections on non-tumorous skeletal conditions, of which there are so many, must be doubted. Scant justice is done to them in the space allowed. Why include rickets, for instance, and dismiss it in three lines? The occasional confusion of scurvy with osteogenic sarcoma is emphasized, but the much more difficult diagnostic problem which may be produced by haemophilia is not mentioned. It is surprising to find that the frequently occurring problem of the diagnosis of simple osteoporosis from the diffuse skeletal rarefaction of some cases of myelomatosis and carcinomatosis is not considered.

On the whole, the chemical and radiological features of the various conditions are clearly and fully presented, with many valuable practical suggestions regarding the management. The book should prove of great value as a textbook for those studying for higher degrees in medicine, surgery, radiology and perhaps even pathology. It will be very useful as a reference book to those of us who deal with any aspect of bone disease. W.P.U.J.

CHIRURGIE EN BEJAARDE PERSONE

Management of the Aged Surgical Patient. By Sidney E. Ziffren, M.D. With chapters on *Anesthesia* by Stuart C. Cullen, M.D. and *Urology* by Rubin H. Flocks, M.D. and a discussion of *The Cardiac and Diabetic Patient Facing Emergency Operation* by Raymond F. Sheets, M.D. Pp. 219. 30 figures. $7.50 Chicago: The Year Book Publishers, Inc. 1960.

Na 'n oorsig van die algemene probleme van die bejaarde pasiënt wat chirurgiese behandeling nodig het, gaan die skrywer oor tot meer spesifieke oorwegings van die voorbereiding van dié soort pasiënte vir operasie—met nadruk op die belang van korreksie van bloedarmoede en elektrolitiese steurings.

Die operasie, na-operatiewe tydperk, en die komplikasies word in die algemeen bespreek, asook die akute buiktoestande by oumense. Dan volg daar 'n hoofstuk oor 'n verskeidenheid van toestande, insluitende die arteriosklerotiese bloedvatsiektes.

In 'n hoofstuk oor die probleme van die diabetiese pasiënt en die pasiënt met hartversaking wat noodoperasies moet ondergaan, deur mede-skrywer R. F. Sheets, word 'n oorsig gegee oor die spesiale voorsorgsmaatreëls wat hier geneem moet word. In 'n hoofstuk oor narkose deur S. C. Cullen word hierdie belangrike onderwerp, tesame met die voorbereiding en naoperatiewe sorg, uit die narkose-oogpunt bespreek.

In 'n hoofstuk oor troumatiese letsels word die aandag meestal gewy aan ortopediese probleme, en ten slotte bespreek R. H. Flocks die spesiale urologiese probleme by ou pasiënte.

Alhoewel hierdie 'n betreklike beknopte samevatting van die onderwerp is, dien dit nogtans as 'n baie waardevolle leidraad by die oorweging van die chirurgiese behandeling van die bejaarde pasiënt, en die skrywer sluit 'n lang lys van verwysings na oorspronklike publikasies in waarna die leser kan verwys vir meer informasie oor besondere aspekte van die onderwerp. J.J.W.vZ.

GYNAECOLOGY

Synopsis of Gynecology. 5th Edition. By R. J. Crossen, M.D., D. W. Beacham, M.D. and W. D. Beacham, M.D. Pp. 340. 106 illustrations. South African price: R5.52½. St. Louis: C. V. Mosby Co. 1960.

Another useful little book of the American 'synopsis' series; it is attractively bound and produced.

The content is presented in a practical way, the introductory chapters being concerned with anatomy and physiology, and gynaecological examination and diagnosis. The succeeding chapters follow in anatomical sequence.

The volume should be of practical value and should prove useful to students and general practitioners for rapid reference. P.A.T.

BRIEWERUBRIEK : CORRESPONDENCE

TREATMENT OF SHARK ATTACK

To the Editor: It was not my purpose to enter into an altercation in the pages of the *Journal*, but Dr. Campbell's[1] immoderate and presumptuous remarks in answer to valid criticism make it difficult to return to a detached plane of discussion. While Dr. Campbell and his colleagues are to be congratulated on their successful management of 3 cases in 1960, I feel that treatment likely to give the greatest benefit to the greatest number of cases should be recommended. I cannot understand his disinclination to apply methods which are acceptable to acknowledged experts in traumatology. Although he fails to add much information about his experiences before and after adopting the new régime, we are obliged to accept his dicta on the treatment of shark attack unless we personally have had the experience of treating a shark-bite. We would then be in the position of having regional experts in shark-bite, lion-bite or crocodile-bite each denying that 'overseas methods of treating shock' were applicable to his problems. Let me suggest that Dr. Campbell stifle his obvious affront at being contradicted, spend a little more time and thought in the library, and acquaint himself with other opinions on the management of massive injuries in field conditions.

To those whose everyday experience and interest has been resuscitation and the treatment of blood loss, both in surgical and traumatic injury, required reading has been the tremendous work of the American team in Korea, of the Birmingham Accident Hospital Staff, and of the British Medical Corps in action in Cyprus and Korea. Surgically, it matters little whether a man loses a limb to a shark, to a land-mine, or in a machine-shop. The first consideration is arrest of haemorrhage and replacement of blood loss, and unless Dr. Campbell can convince us that special considerations apply to the shock and trauma of shark injuries, we insist that similar methods of treatment must apply. It is also argued that conditions on the Natal coast are so exceptional that a particular form of treatment is applicable there. These beaches are nearly all within easy reach of a small hospital and are readily accessible from a good road. In the only case reported by Dr. Campbell in detail,[2] it took only 45 minutes to reach hospital in Durban, a distance of 16 miles. What makes conditions on the Natal coast so exceptional that special methods should apply to these victims that do not apply to injured persons in other situations?

The argument boils down to three points which I criticized in the treatment advocated by Dr. Campbell:

1. *Morphine.* He advocated morphine, gr. ¼ - ½, in every case whether pain was present or not. Pain is not often a feature of severe injury (in Dr. Campbell's reported case[2] it was emphasized that despite very severe injuries the patient at no time experienced any pain). We repeat that morphine must be given with care and discretion in oligaemic shock. Watts, in a 1960 Hunterian lecture[3] on missile injuries in Cyprus, emphasized that one of the commonest faults in first aid was oversedation. He repeated what we have commonly found in civilian practice—that morphine may produce severe respiratory depression and may prejudice the chances of recovery. Restlessness usually results from cerebral anoxia following hypotension and anaemia, and responds to administration of blood. Morphine-induced respiratory depression makes general anaesthesia hazardous. Dr. Campbell must give his reasons for believing that large doses of morphine helped to save his patients' lives for, in fact, from the details available we do not know which paients had intravenous therapy before transportation to hospital. Surely he will not argue the disproved theory that morphine is specific in its action against shock? Why then advocate as a routine measure large doses of morphine?

2. and 3. *Transfer to hospital or beach resuscitation.* The most important first-aid measure in these injuries is to stop the bleeding. No 'new' or 'original' principle is involved, but it deserves greater emphasis. I criticize the advice to apply tourniquets and stress that pressure applied by firm packing of the wound and bandaging is the most effective method of stopping bleeding. Once applied and seen to be effective, such packs must not be taken off until the patient is on the operation table and has been adequately resuscitated. I repeat my criticism of Dr. Campbell's advocacy o leaving patients lying quietly on the beach for up to one hour, because, even if it is practicable for the transfusion of plasma or blood to be begun within that period, it will be inadequate in severe cases, and unnecessary in less serious injuries. It can only delay the transfusion of the large amount of blood which may be needed to save life, and which can best be done in hospital. Every hospital should hold blood in a bank or have a donor panel organized.

Transfer to hospital offers a better chance of success than attempts to resuscitate patients on the beach. Delay without blood transfusion is dangerous and irrational. The few facts published by Dr. Campbell do not support his thesis that sedation and delay improved patients, nor that transfer to hospital caused the death of those who died.

I recommend him to read the recently published Ruscoe Clarke Memorial lecture by Lt.-General Sir Alexander Drummond,[4] Director General, Army Medical Services, on the practice recommended for the British Army, which is based on extensive experience of severe injuries in actions as far spread as Malaya and the Mediterranean. This confirms each of the opinions which I have previously expressed.

Despite Dr. Campbell's protestations that his mind and those of his colleagues are made up and will not be changed, I urge him to temper his enthusiasm by comparing his own experience with that of others in other fields of traumatology and permit others with a critical faculty the right to compare his work and conclusions with their own views.

318 Medical Centre
Heerengracht
Cape Town
14 June 1961

D. M. Jowell

1. Correspondence (1961): S. Afr. Med. J., 35, 462.
2. Campbell, G. D., Davies, D. H. and Copley, A. C. (1960): Med. Proc., 6, 612.
3. Watts, J. C. (1961): Ann. Roy. Coll. Surg. Engl., 27, 125.
4. Drummond, A. (1961): J. Roy. Army Med. Cps, 107, 35.

GENERAL PRACTICE IN SOUTH AFRICA

To the Editor: I recently overheard a very nice compliment paid to general practice in South Africa. An American, who has been in this country for two years and has travelled widely, remarked that it was the only country he had visited in which real medicine was still practised. He referred in particular to general practice.

He also remarked that, in view of the general income level, the remuneration received by the general practitioner was among the lowest he had encountered.

Philip H. Dalgleish

Hill Crest
Natal
8 June 1961

GASTRODUODENAL HAEMORRHAGE

To the Editor: I read Mr. Kane's article[1] on his experiences in the management of gastroduodenal haemorrhage with interest. I am curious about his statement in the summary: '2. The claims for soluble aspirin are contested'. In carefully reading the article he nowhere provides evidence that any of his cases of haemorrhage were in fact precipitated by taking soluble aspirin.

Even in a preliminary report I do not think that such unsupported remarks should be made, especially since there are several articles in which ordinary aspirin and soluble aspirin have been carefully compared clinically, to the advantage of the latter.

If Mr. Kane has contrary evidence I look forward to seeing this clearly stated.

Pincus Catzel

Klerksdorp
Transvaal
7 June 1961

1. Kane, G. J. (1961): S. Afr. Med. J., 35, 454.

INCREASED MEDICAL AID FEES

To the Editor: The Editor's assumption that the suggestions in my letter[1] on increased medical aid fees would bring to an end the operation of medical aid societies is, in my view, incorrect. In any case this is certainly not what was intended, since I am a firm believer in medical aid societies themselves, but I maintain that they must never be in a position to dictate the terms and conditions on which they are to enjoy a preferential tariff.

I have pointed out that general practitioners in Johannesburg are far from satisfied with the recent six-point agreement, whereas the schedule of alterations might suit other general practitioners in various parts of the country. It is therefore important that the voice of the general practitioners in Johannesburg be heard in the Councils of our Association, otherwise they will be forced to enunciate their own schedule of private fees—a step which will compel medical aid societies to fall into line.

A. D. Bensusan

7 St. Paul's Road
Houghton
Johannesburg
6 June 1961

1. Correspondence (1961): S. Afr. Med. J., 35, 464.

[We did not intend saying that Dr. Bensusan's suggestions 'would bring to an end the operation of medical aid societies', but that, if it is accepted in principle (among other things) to raise the preferential tariff of fees in certain urban areas to the level of 'treble the present country fee', the medical aid societies will find it impossible to meet the demands made on them and will *for that reason* have to cease operating—Editor.]

TRANQUILLIZERS

To the Editor: Allow me to contradict Dr. W. Leigh's[1] statement that addiction to 'noludar' is unknown. Among my addicts I recall at least ten noludar addicts, some of them, including two doctors, now dead. It is precisely its mildness that makes this drug so popular with addicts and therefore so dangerous. Twenty or thirty tablets spread throughout the day will not knock addicts out completely, as in the case of barbiturates, but will produce the 'delightful' dream-like state they long for. I have learned to spot the noludar-drugged patient at a glance: the slow, ghost-like incoordinated gait and movements, the slow, slurred speech, the inability to focus the eyes, the incoordinated efforts to strike a match, the tell-tale burned holes in the bed clothes.

Whatever distinction Dr. Leigh would like to make between tranquillizers and sedatives to prove his point, these two groups of drugs are similar as far as addiction is concerned. The classical dependence and the withdrawal syndrome are found in the case of meprobamate as well as noludar.

I personally see neither 'interference' nor 'a slanting attack on the medical profession' in Miss Marwick's[2] remarks and her warning against the abuse of drugs. Dr. Leigh's rudeness to this distinguished member of the nursing profession is unwarranted and stems, I suspect, from his commercial interest in this particular drug.

S. Livni

1202 Rand Central
Jeppe Street
Johannesburg
6 June 1961

1. Correspondence (1961): S. Afr. Med. J., 35, 463.
2. *Idem* (1961): *Ibid.*, 35, 400.

THE DANGERS OF EARLY WEANING

To the Editor: Iatrogenic disease is often in the news. In this hospital we encounter it not infrequently: a marasmic, ricketty infant, aged a year or more, is admitted in a near-terminal state precipitated by an acute gastro-enteritis or bronchopneumonia. The baby has been weaned since one or two months of age. We ask why, and we view with horror the dirt-encrusted teat and bottle protruding from the mother's skirt pocket. The inevitable answer is: 'I took my baby to a private doctor in town who told me my milk was bad for the baby and advised me to take it off the breast'. No further feeding instructions are given. The doctors concerned have been both European and non-European. Whilst in town, the mother attempts feeding with milk or other infant food. On her return to the Reserves, where no fresh milk is available, a tin of powdered milk is eked out to last as long as possible by dilution to homeopathic proportions. The infant's failure to thrive is attributed in ignorance to everything but the true cause. The mother is now convinced that her milk is poisoned and determines to wean the next baby immediately on birth and forestall the wise physician.

While an enlightened African mother in town may successfully rear her baby on a bottle, I am sure that all who work in Bantu areas will agree that premature weaning under primitive conditions spells a death sentence for the infant. This has been amply borne out by the revolution in the prognosis for babies born to tuberculous mothers; while previously separation of the African baby from its mother meant almost certain infant death, the present régime of continued breastfeeding and prophylactic administration of isoniazid from birth (Dormer) has resulted in thriving and contented babies.

In our experience in this area, every effort should be made to preserve the traditional method of breastfeeding. Many infants are successfully reared on only one breast, following a unilateral breast abscess. Complementary milk-feeds given by teaspoon will tide a tiny infant over the initial breast-milk deficit, so often encountered in twin-births; there is sometimes a rewarding increase in the breast-milk supply in the later months and babies continue to gain well on breast-feeds and porridge only—despite the cessation of complementary milk-feeds.

Prolonged breast-feeding of African infants, whatever its drawbacks, has much to recommend it, especially in rural areas. Weaning in the first few months should be sternly discouraged, more so if the mother intends returning to her home in the Reserves. I have been encouraged by the Medical Superintendent and my colleagues to bring this matter to the notice of the profession.

Edith Waldmann
M.O., Children's Ward
Jane Furse Memorial Hospital (Anglican Mission)
Sekhukhuniland
Via Middelburg, Tvl.
31 May 1961

ENDOTRACHEAL ANAESTHESIA

To the Editor: A simple and trusting reader of the *Journal* might be forgiven if he imagined that an article entitled 'Fatal and near-fatal accidents associated with endotracheal anaesthesia,'[1] would in fact throw some light on some of the possible complications of endotracheal anaesthesia. Not a bit of it; and the shrewd reader will get a clue from the name of the author, Dr. C. S. Jones, and realize that the curious and inapposite title is just an occasion for a continuation of his attack on the use of relaxants in anaesthesia.

Let it be stated forthwith that the method of using a short-acting relaxant for intubation is a well-tried and established technique all over the world, but Private Jones would have us believe that the rest of the army is out of step, whilst he marches on alone in sublime confidence, thanking God (or Nature) that he is not as other men are.

We can all be wrong, and I am willing to change my mind and practice if I can be shown to be wrong. However, the cases reported by Dr. Jones and the arguments used do not convince me that the use of a relaxant for intubation had anything to do with the appalling results he quotes. Incidentally, Dr. Jones was not present during the administration of any of the cases he quotes, and the sketchy reports are therefore only hearsay evidence. I intend to deal with each case in turn.

Case A. The relaxant used is not named. I wonder whether during the ensuing 20 minutes there was not some hypoventilation and hypoxia, resulting in cardiac arrest. In any event, cardiac massage and adequate oxygenation does not seem to have been effected early enough, because the patient never recovered. No cause for cardiac arrest is the usual finding of an autopsy in such cases.

Case B. This child was mentally retarded. It is a well-known fact that such children often have other unsuspected congenital abnormalities. Such may have been the case, and the absence of an autopsy report means that it cannot be excluded. Incidentally, as this was a death from unnatural causes, there is a statutory obligation to perform an autopsy, and one must ask why no autopsy report is available. One does not know for how long the heart had stopped before the surgeon (not the anaesthetist) noticed

abnormal colour. As cardiac massage failed to restart the heart, it may have been stopped for too long already. In any case, there seems to be no reason for blaming the relaxant, again unnamed, any more than the thiopentone, or the premedication, or any other factor one's particular hobby horse drives one to blame.

Case C. Again our unnamed relaxant; again it may still have been working and the patient allowed to hypoventilate and become anoxic. In the face-down position used for laminectomy, the effect of any hypoventilation would be aggravated. Cardiac arrest is said to have occurred when the incision was made. Is it not more likely that it had already occurred during the positioning and towelling and was only recognized as soon as the incision revealed a bloodless field? Again obviously, the restarting of the heart and re-oxygenation of the brain were too long delayed for the patient to recover consciousness.

Case D. Again we are not told what the relaxant was. It is not clear why it was thought so necessary to intubate the patient. After direct intubation under vision had failed (and what anoxia did the patient suffer during the unsuccessful attempts?) blind intubation is presumed to have been successful. But the patient remained cyanotic for 'a considerable period'—why? If the endotracheal nasal tube were truly in the trachea there can be no excuse for prolonged cyanosis. Obviously, either the tube was not correctly placed or insufficient oxygen was delivered down it. Prolonged anoxia is enough to account for the sequelae and there is no need to try to blame the relaxant.

Case E. This patient is the only one in the series who received a known dose of a named relaxant, 80 mg. of 'flaxedil' to wit. This dose in a healthy male of 38 years would probably not even cause temporary apnoea, in fact we are told later that hyperpnoea developed. This patient received trichlorethylene, both before intubation and during maintenance. The so-called hyperpnoea was probably tachypnoea, a sign of overdosage with trichlorethylene, which drug is well known to cause cardiac arrhythmias. Next, when the pulse was absent and heart sounds could not be heard, 4 - 5 precious minutes (longer than the brain can stand anoxia) were wasted giving noradrenaline. Taking into account the unforgivable delay in performing cardiac massage, I am surprised that the patient survived, but not a bit surprised that he is demented, and the relaxant most certainly had nothing whatever to do with the outcome.

Case F. Once again, the relaxant is not identified. There appears to have been no trouble with the anaesthetic during the operation. Six weeks unconsciousness, followed by personality changes and death, are all attributed to one dose of relaxant which produced no evident complications within the first few hours, by which time the known (not Dr. Jones' hypothetical) effects of any relaxant would have been completely eliminated. Dr. Jones is so blinded by his own unique pet aversion that he cannot see any possibility that either a deterioration of the patient's original lesion, already sufficiently serious to warrant craniotomy (or dare we suggest, the operation itself) might have been the cause of the unfortunate postoperative course'.

Thus, not one of the examples referred to by Dr. Jones is even suggestive, let alone convincing, of any untoward result of using the relaxant. Further, Dr. Jones cannot accept that ventilation with pure oxygen can maintain oxygenation and remove carbon dioxide, 'better than the patient himself, when anaesthetized in an old-fashioned way'. Experiments have been done on many occasions using partial-pressure studies in alveolar air, and arterial gaseous tensions, and it is not necessary for a practising anaesthetist to repeat these every time he uses the technique. But I am willing to demonstrate that both adequate oxygenation and efficient carbon-dioxide removal can be effected clinically by intermittent positive-pressure respiration on subjects paralysed by relaxants, whereas a patient anaesthetized in an old-fashioned way when sufficiently deep for atraumatic oral intubation under laryngoscopic vision will, while probably adequately oxygenated, most certainly have his respiration sufficiently depressed to result in some carbon-dioxide retention. That is when Dr. Jones should start using his partial pressure and gaseous tension studies.

The publication of this article, which may give those ignorant of the true facts an impression that modern anaesthetic practice is dangerous and irresponsible, is particularly unfortunate at this time when Dr. Jones has just left South Africa to take up a position in Australia. Perhaps this wandering prophet may find honour in his new country. Dr. Jones has shown his ignorance of the value of the routine use of relaxants in modern anaesthesia, and as a result the publication of his article may well create unwarranted fears in the minds of both the public and the profession. It could also create unnecessary and unwarranted difficulties in inquest inquiries in cases in which relaxants have been used, however momentarily.

His paper, in view of the publicity it must necessarily obtain, could be highly prejudical to the sound practice of surgery and anaesthetics.

F. W. Roberts

Jeppe Street
Johannesburg
5 June 1961

1. Jones, C. S. (1961): S. Afr. Med. J., 35, 421.

ENDOTRACHEAL ANAESTHESIA

To the Editor: Dr. C. S. Jones' article in the *Journal* of 27 May 1961[1] cannot go unchallenged. In my opinion this paper is grossly misleading, unscientific, and unexpected from the Head of a Department of Anaesthetics.

Dr. Jones has reported 6 cases of cardiac arrest which he attributes to passage of an endotracheal tube, using muscular relaxants. This inference is based not on scientific data, but on pure assumption. He states that 'it is extremely difficult to resist the conclusion that death or disablement was caused either by anaesthetic agents used, or anaesthetic techniques, or both', although in his report there is absolutely nothing to suggest this whatsoever, except in case D in which there was a problem associated with intubation. His autopsy findings are either not available or show nothing significant to substantiate this suggestion. Important facts, which I personally feel should have some bearing on the outcome, are contained in the case histories which Dr. Jones sees fit to ignore in a most unscientific manner, e.g.

Case A—old depressed fracture of skull.

Case B—mental disorder in a child where air studies were advised.

Case C—spinal pathology.

Case D—a full pre-operative history is not given, nor is a full postoperative investigation done.

Case E—spinal lesion, patient apprehensive, but no sedation given. Why? Although this is beside the point, I should like to add that the management of case E was, to my mind, contrary to physiological principles, since noradrenaline was administered by intravenous infusion for 4 - 5 minutes after cardiac arrest. I think that the fact that the patient is still alive is quite remarkable under the circumstances.

Case F—headaches. The symptoms severe enough to warrant opening the posterior fossa. Dr. Jones sees fit to ignore completely the history and the procedure in the production of postoperative problems, yet jumps hastily to the conclusion that the muscle relaxant used to intubate the patient, probably hours before, is the causative factor in the long period of postoperative unconsciousness and other cerebral problems that followed. I feel most emphatically that this is unscientific and very misleading.

The statement that these cases reinforce the contention that the need for endotracheal intubation does not constitute an indication for the use of muscle-relaxing drugs is, to my mind, complete nonsense. The use of the relaxants in this sphere is a widely practised technique which is universally accepted.

Finally, I would respectfully submit that this article, which I have no doubt will receive wide publicity, will do nothing but harm to the practice of anaesthesia in this country, and I feel that its publication is an error in the good judgement of the Editorial staff.*

Michael S. Kramer

Jeppe Street
Johannesburg
5 June 1961

1. Jones, C. S. (1961): S. Afr. Med. J., 35, 421.

[*It would have been unreasonable to have refused to publish the views of the Head of a department in a teaching hospital, without having given his colleagues the opportunity of examining his point of view in the spirit of critical assessment.

In encouraging dignified polemics on a scientific level, which might lead us *not* 'to come out by the same door as in (we) went', we are, in fact, rendering a service—Editor.]